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ORAL TUBERCULOSIS.

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In taking up disease of any organ, cavity, or part of the body, it must be considered in relation to other organs affected by the same disease, or in relation to the frequency of affection of the given part by other disease.

In considering this subject it is difficult to separate cases where contiguous organs are involved, therefore, some cases of tonsillar and pharyngeal tuberculosis must be included, but only those where, at the same time, tuberculosis of some part of the oral mucosa has been found.

ETIOLOGY.

All authors divide infections first into primary and secondary, but some authors, as Lake, class secondary cases in the primary class, simply because they consider any infection primary unless there is pulmonary involvement. All cases will be considered as primary if the lesion described is the first demonstrable tuberculous focus in the body.

That the mucous membrane of the oral cavity is not attacked by disease with greater frequency has been marveled for ages. The theories advanced to explain this apparent immunity are

many: First, we have the rich blood supply to the parts; second, thickened layer of epithelium; third, the constant movement of the tongue and cheeks, keeping the parts clean; fourth, the supposed germicidal action of the salivary secretions; fifth, mechanical protection offered by mucus; sixth, protective action of various bacteria in being antagonistic to various other organisms.

It must be admitted that in any case where one of the protective influences is lacking, infection may take place. Realizing the varied conditions under which we live, and with diseased conditions surrounding this cavity on all sides, it is only to be wondered that infection does not more often occur. Considering the number of wounds of the oral mucosa which take place in the healthy subject from biting the tongue, cheeks or lips alone, and which are apparently healed within twenty-four to forty-eight hours with no sign of infection, we should reasonably expect transplanting of infectious material from one organ to another in the individual with diseased respiratory tract. This is especially to be expected in connection with the mouth, as it is used as an accessory respiratory organ in many cases, and because of the infectious material coughed from the lungs or aspirated from the nasopharynx passing over its surfaces.

Mouth infection from the gonococcus is exceedingly rare, although the conjunctiva is frequently involved; in a few cases the oral cavity of the newborn has been found affected; there is only one case of infection of an adult known to the author, which was reported by Cook.

The different lesions due to the tubercle bacillus show, the author believes, different modes of infection, just as we find in the laryngeal and nasal manifestations of this disease.

Tuberculosis of the mouth manifests itself in the following form:

1. Nodes and nodules.
2. Tuberculoma or tuberculous gumma.
3. Tuberculous abscess.
4. Tuberculous papilloma.
5. Tuberculous fissure.
6. Tuberculous ulcer Lupus ulcers.
 Tuberculous ulcers.
7. Tuberculosis of salivary glands.

Infection in primary cases, and it is at present an established fact that such may occur, is either through the intact epithelium, solution of its continuity, or through a cavity in a tooth in which the pulp has been exposed by decay.

That bacteria can be made to pass through the intact epithelium of the mouth, although with difficulty, certainly much more than through the tonsil, has been proved by Dmochowitz, Wood, Grocier and others; and through the larynx by Orth and E. Frankel. However, this probably very rarely occurs, as injuries to the mucous membrane of mechanical origin, as well as chemical and thermal injuries to the epithelium, prepare the way so very frequently that it is not necessary to assume the passage of bacteria through the intact epithelium. Cracks and fissures of the tongue and lips, from drying of tissues, in mouth breathing, predispose to both primary and secondary infection, and they are a special factor in the secondary form, as a large number of patients suffering from pulmonary tuberculosis are mouth breathers. Furthermore, decay of the teeth, with lodging of débris at the gum margin which may work below the epithelium by the side of the tooth, carrying bacteria with it, is a frequent occurrence.

All these contribute to primary infection, which is very rare when compared with the number of secondary cases found.

Adami and Nichols seem to regard hematogenic infection, in cases of pulmonary tuberculosis, as proved. They refer to it as follows: "It used to be, and in many quarters is still, accepted by clinicians and pathologists, that tuberculosis of the lungs is bronchogenic as a rule. It should be mentioned, however, that by certain observers, notably Ribbert, Aufrecht, and Baumgarten, a more or less successful attempt has been made to diagnose this. Ribbert, while not denying absolutely that infection may take place by inhalation, believes that pulmonary tuberculosis is usually hematogenic in the sense that the peribronchial glands are infected through aerial transmission by way of the buccal mucosa, and that when they break down the products of the destructive inflammation are discharged into the blood and so reach the lung. Baumgarten goes still further and holds that the glands are also invaded hematogenously. The experience of Aufrecht would seem to prove that it is impossible for bacilli to reach the terminal bronchioles and alveoli through inhalation, and postmortem evidence

also supports this contention. It would seem probable that we would have to give up the view that the bronchi are the first structures to be attacked, and adopt a modified inhalation theory somewhat similar to Ribbert's, admitting an infection through the blood or lymph stream from the mouth and nose and upper respiratory passages." These authors also mention the work of Ravel, which is along the line of Wood, Robertson and Goodale, that ingested tuberculous material may be found in the tonsils and bronchial glands.

If the contentions of these investigators are true, why do we not have more cases of primary tuberculosis of the nose and mouth if infection takes place through the mucosa of these cavities, and why do we not also find tubercle bacilli in the blood? If infection through the buccal mucosa without injury to the epithelium reaches the lungs through the blood stream, why should the larynx, esophagus, pharynx and brain, or even the tissues of the neck, not be involved instead of the lungs?

The investigations of Cornet, by inoculation of nearly three thousand guinea pigs and rabbits in different parts of their bodies, show that the first tuberculous changes are found on the spot of inoculation, or in the lymphatic glands draining that region. These experiments have resisted practically all contradiction, as most other experimenters have found the same to be true. A very few exceptions to this rule might be explained on the ground of faults in technic, or in anomalies of the lymphatic system, but these exceptions are so few that they can practically be ignored.

The mode of infection in secondary tuberculosis of the oral cavity differs but little from that of infection of the larynx, pharynx or tonsil. It is highly probable that the nodular infections, the tuberculomata, so-called, and tuberculous abscesses, are of either lymphatic or hematogenous origin, as they all make their appearance in about the same way. First, a rounded eminence, which either gradually enlarges or remains about the same size for months or years, when it shows a slight ulceration on the eminence. This gradually breaks down, allowing the discharge of pus, or shows a granulomatous condition which quickly disintegrates and exposes a cavity with undermined edges.

On section, the wall of the abscess does not differ from

that of a tuberculous abscess elsewhere. It contains miliary tubercles in which are found giant cells and tubercle bacilli. The pus may not contain enough tubercle bacilli to be demonstrable, but on inoculation of guinea pig, death from tuberculosis will be produced.

Nodes appear on the tongue, according to Butlin, as small tubercles from one to four millimeters in diameter; too small to attract attention except where the tongue is one of the organs included in an active miliary outbreak. The nodules, or small yellowish lumps, are caused by the fusing of several of these tubercles, which may be as small as a pin point, or become as large as a hazelnut before breaking down. In these cases they resemble tuberculoma. Butlin considers the following as points for diagnosis: The absence of signs of past syphilis and the failure of antisyphilitic remedies, evidence of tubercle in patient, lupus or glandular scars, tubercle bacilli in sputum, if patient has pulmonary involvement.

Attention, however, must be called to the fact that it is difficult to differentiate between syphilitic scars and those produced by lupus. The Wassermann reaction in syphilitic cases relieves us of the necessity as well as of the waste of time incident to the therapeutic test.

Tuberculoma is a term which, with our present knowledge of tuberculosis, should be eliminated, as it means nothing more or less than a tubercle which is formed because of fusing of many small tubercles. It may go on to abscess, or may undergo practical calcification; or, if it ceases to grow, we may have extensive fibroid changes in the capsule with caseous degeneration, and the supposed tumors remain for years without change.

These tubercles may be single and may remain for years without discomfort except for the slight enlargement.

Notable in this connection is the case of Richardson, a young lady, nineteen years of age, who had a tumor mass in the middle of the dorsum of the tongue. The patient believed this condition had been present since childhood. The case had been under Richardson's observation for about one year, when this mass became suddenly active, causing excessive pain in the throat and ears; this was relieved by the evacuation of a thick caseous pus.

The cases of de Brun, de Bois Nor, Thorner, Kyle, and Goselin and Fano come under the same classification.

On the other hand, multiple tumor-like masses are found less frequently, either in the tongue or elsewhere in the mouth, although nearly all cases show tubercle in the surrounding tissue; but these are small, while those that are called nodules are represented by such cases as that reported by Barth. In his case there were a large number of nodules giving the so-called fossilated appearance and varying in size, several of which later broke down, forming ulcers.

According to latest investigation it has been found that when tubercle bacilli introduced into the lymph or blood circulation lodge on the wall, they are almost immediately taken up by the endothelial cells, causing them to become swollen. It is to be seen by this that the first reaction on the part of the tissue is a cell growth and not a cell destruction.

These cells are what have been known as epithelioid, and, as takes place when any foreign substance is introduced into the tissues, lymphocytes collect around these so-called epithelioid cells, at first only polynuclear, but later lymphocytes.

From this proliferation and swelling the capillary involved becomes occluded, and the surrounding vessels will later become occluded in a similar way, so that if the process begins in a vessel, it will later become an intravascular formation. It is easy to be seen that the first stage of the formation of the tubercle is a cell proliferation. One or more of these endothelial cells may grow to a large size with a diminution of the nucleus, or several may fuse to form a giant cell. Which of these takes place more frequently is a matter on which pathologists differ.

It has been proved that dead tubercle bacilli will produce all of these early changes. This may be taken to mean that these stages are induced by the gradual diffusion of the intracellular toxins.

It is also believed that cell proliferation may be produced by the toxins liberated by living bacilli, or by those liberated by tubercle bacilli which may be killed by the lymphocytes or endothelial cells. It has been suggested by Prudden and Hoenpfl that this cell proliferation may be produced by the toxins acting in a dilute solution in the tissues, which accounts for the first stages being produced by the introduction of dead

bacilli, while the living organism, producing the toxin in larger quantities and more concentrated, will finally occasion necrosis. This also explains the poor staining qualities of the giant cell as one side of it begins to necrose. It is also peculiarly significant that the tubercle bacilli are found on the side where necrosis is taking place, although broken down bacilli may be found in the central portion. The nuclei in the giant cell are found arranged in the form of a crescent, and surround this necrosed mass.

The tubercle early presents a giant cell in the center, surrounded by larger epithelioid cells with lymphocytes interspersed. These are found as typical round cells in subacute and chronic inflammatory conditions, at other times polymorphonuclear leucocytes. Surrounding this may be found some capillaries showing dilatation, but not markedly so, as is found in acute inflammation.

Necrosis of this area progresses in proportion to the rapidity of the growth and multiplication of the bacilli, and we have an increase of volume of this central area, and necrosis of the caseous type. As further progression takes place the cells lose their power of taking the stain, and lose their outline, besides undergoing granular and fatty degeneration. Around this caseous mass filling the center we have arranged younger giant cells, and these increase in number, enlarging the ring as the central areas increase in size; and so this growth may go on, especially in the tongue, until the mass is not only visible to the naked eye, but also becomes of such proportions as to be compared in size with beans or nuts. Attention is also directed to the fact that the bacilli are not only intracellular, but are found interstitial as well.

If any bacilli escape, they are either destroyed by the endothelial cells or leucocytes, or are carried by the latter to other parts of the body where they come to rest; most probably in the lymph spaces, where they form new foci for dissemination in acute cases, or new tubercle in chronic. If these tubercles in their continued growth come in contact with the wall of a large blood vessel bronchus or surface, we may have necrosis of the wall and the formation of an ulcer.

Further, tubercles may become calcified, and the procedure is somewhat similar to encapsulation in any chronic abscess condition. The tissues become somewhat accustomed to the

toxins and the bacilli are probably weakened so that the tissue adapts itself to the existence of the foreign substance within it, then the fibroblasts, which are found in the periphery of the tubercle, develop into fixed connective cells, forming a thick capsule.

It has also been proven that a tubercle may be absorbed, as is seen in peritoneal tuberculosis, and in some cases of joint tuberculosis.

What effect mixed infection may have upon tuberculosis is no longer a question, except as it affects different tissues. We know that the epithelial tissue proper is not affected by tuberculosis; and Ely claims that connective tissue is never affected by tuberculosis unless we also have infection with other organisms. Many pathologists do not agree with this theory, but from the standpoint of the stomatologist it makes very little difference, as we probably always have mixed infection. Cases of secondary hematogenous infection from the pulmonary lesion may possibly prove exceptions, as we may also have other organisms associated with the tubercle bacillus.

It is well known that cases with mixed infection are more rapidly fatal, or are not as amenable to treatment as those in which this is a pure infection. This may account for the old idea that the nearer the exterior of the body the worse the prognosis in tuberculosis. If this were entirely true, as it is undoubtedly to a certain extent, but for a different reason than given by the older authors, it may be due to the more frequent occurrence of mixed infection. However, the patient is weakened by disease, the tissues of the mouth and pharynx being deprived of their rich blood supply, two foci of tubercle develop, together with the interference with nutrition caused by the difficulty in either mastication or deglutition, or both, and thus seriously handicapped the patient comes frequently to an early end. This applies only to cases of secondary localization of the disease in the mouth. Until the last few years a bad prognosis was given every case of laryngeal tuberculosis.

The fact that many cases of oral tuberculosis break down after healing does not prove the tubercle to be any different than in the larynx, lungs, or any other part of the body. The least lowering of vitality, either in a general way or locally,

tends to favor development of bacilli already present. It has been proven in cases of quiescent tubercles that the bacilli remain alive for years, since they prove fatal to guinea pigs. In still other cases we may have sterile pus.

There is very little difference of opinion regarding the location of the lesions of tuberculosis in the mouth, although some modern textbooks, notably McFarland's Pathology, give the base of the tongue as the most frequent location of tuberculous ulcers.

The writer's investigations seem to prove that the parts are affected in the following order of frequency:

1. Tongue, tip.
2. Tongue, border and floor of mouth.
3. Palate, soft, anterior pillar, and uvula.
4. Tongue, dorsum and base.
5. Lower jaw and gums.
6. Upper jaw and gums.
7. Lips.
8. Palate, hard.
9. Salivary glands.

This corresponds closely with that given by other investigators. However, a decision is very difficult to obtain, inasmuch as many reporters fail to state explicitly the part affected. This may be due either to unfamiliarity with the nomenclature applied to the mouth, or to carelessness. We have, at times, appearing in the mouth and more frequently upon the tongue, what the patient describes as a blister, and it looks very much like one if observed before it breaks down. This, apparently, is a small tuberculous abscess, which breaks down and liquefies suddenly so that fluid is seen under the mucosa. It is probably due to mixed infection, the secondary organism gaining entrance to the tubercle through the epithelium.

The writer has observed this lesion in two cases, Nos. 7 and 14. One of these died within ten days after the appearance of the lesion, and the other lived about two months. It first breaks down in the middle as a fissure, as these blisters are always oblong, and leaves an ulcer with overhanging edges but with a soft base. The edges show no tendency to slough, but the overhanging portion, if on the tongue, is very smooth,

as the papillæ have disappeared. The fissure thus described differs from that reported by Butlin.

Ulcers may also appear as fissures, which, in the early stages, may be entirely overlooked, resulting from infection taking place either from the sputum or through the blood or lymph streams. They are most frequently found on the sides or tip of tongue (Fig. VIII), although they may also appear upon the dorsum and at the corner of the mouth. They may or may not be found in a fissure of a previously fissured tongue, and, although the edges are usually soft, they may be indurated. Butlin describes them as having indurated edges. When the fissure is separated the granulations appear as in a tuberculous ulcer of the surface, but with fewer vascular granulations, and with minute tubercles over the whole surface, interspersed with light reddish areas. The edges slough slowly as a rule, from caseation of the tubercles, leaving ragged edges. These fissures may extend into the muscular substance of the tongue and yet show no surface extension, but not so at the corner of the mouth. Here surface extension similar to ulcers beginning on the surface of the tongue, cheeks, or gums may be shown. Ulcers appearing upon the lips, cheeks, surface, edge or tip of tongue, usually exhibit a reddish base covered with granulation tissue, upon which small whitish tubercles seem to have been sown. The edges are irregular, or what was first termed by La Bole as mouse-eaten, soft, although they may show some induration.

Some authors speak of the sharp-cut appearance of the edge. This is very seldom to be found, although the writer has seen two cases in which the edges could be said to be sharply outlined. The tissues immediately surrounding the ulcer are pale and flabby in appearance, and may show many tubercles scattered over the surface. The tendency is to spread laterally but not deeply, but some cases also show destruction of muscular tissue.

Ulcers of the gums begin at the gingival margin, usually around a tooth having an artificial crown, one having a calculus upon the root, or around a much decayed and broken down tooth. (Cases 2, 5 and 16.) This continues into the mucosa and periosteum, involving the alveolar process, which, in some cases, breaks down slowly, while in others so rapidly that the whole socket becomes involved, causing the teeth to loosen to

such an extent as to become not only a marked annoyance but also very painful, so that removal is necessitated. The process of destruction continues, involving usually the socket of the tooth upon either side, until the angle of the jaw is reached, or a vacant place where a tooth has been extracted. In the first, the palate may become involved, while in the latter the destruction may not jump the space.

Other parts of the mouth may be involved; more frequently the gums of the other jaw, cheeks, lips, and floor of the mouth, in the order named (Cases 2 and 16). The gums of the opposing jaw are affected possibly from direct transference of infection by the tongue or food. In the case of other structures named, it is by direct continuity of the mucous membrane.

Bower's case caused exfoliation of several teeth, and several sequestræ were removed. This patient complained of a great deal of pain referred to the ears; probably either through the auriculotemporal or Jacobson's nerve, as the reporter does not state in what part of the ear the patient located his pain. Both sides of mandible were involved.

Chamberlin reports as cured by local applications the case of a girl, fifteen years of age, showing primary involvement of the buccal surface of the gums of the lower jaw.

Stephen Paget also reports an interesting case of ulcer of the gums which spread to the lip. This patient was a woman, twenty-six years of age, who had pulmonary tuberculosis. The ulcers had been present for three years. They were thoroughly curetted, but returned.

In the case reported by Coolidge, the gums were affected back of the anterior teeth, and ulcer healed after use of electric cautery. Coolidge suggests that infection of the nasal cavity may take place in this way, while Grünwald believed that his case of tuberculous ulcer of the hard palate was infected from the nose.

Walter reports case of male, aged thirty-eight years, in which, as he expresses it, perforation of hard palate exposing maxillary sinus followed the extraction of a tooth. The wall of ulcerated tract was covered with miliary tubercles. Patient died of pulmonary tuberculosis.

Lenzmann saw a woman, aged twenty-six years, with tuberculous ulceration of gums of lower jaw, which spread to cheek and lower lip.

Butlin makes a classification of tuberculous papillomata, which is nothing more than an exaggeration of a fissured ulcer in which the edges seem to hypertrophy. The writer has never seen a case involving the tongue, and only one, a case of Lockard's, with tuberculosis of the soft palate and lower jaw, having a fungoid appearance of the anterior pillar and of the mucosa of cheek. This man was twenty-three years old, and had extensive pulmonary and laryngeal involvement. He died within a few weeks. This case appeared, on first view, like lupus, but was secondary to lung involvement.

The soft palate is involved many times when no other oral structure is affected, mainly by extension from pharyngeal and tonsillar tuberculosis, and usually very late in the disease. Ulceration is almost always very superficial, covered with a grayish film, with the edges poorly defined. It spreads rapidly, covering the whole palate with tubercles in from twenty-four to forty-eight hours.

One of Lockard's cases, seen by the writer in consultation, presented a perfectly clear palate at the time of treatment, but in forty-eight hours it was completely covered with tubercles.

Grocler saw eight cases of perforation of the soft palate: two primary cases, and six secondary associated ulcerations of gums, lips, tongue, tonsils and pharynx. He concludes that the soft palate is affected more frequently than the hard, while syphilis affects the hard, and that the borders of the specific ulcers are more sharply defined.

However, the hard palate may be affected with loss of tissue and go through the same course as the gums, the tubercle first appearing on the mucous membrane. These tubercles break down and form an ulcer, which continues into the periosteal covering and destroys the blood vessels. In this way nutrition is cut off from the part, and sloughing and necrosis take place.

¹The cases of Coolidge, Walter, Grünwald, and case 2 of the writer's, are examples of ulceration of the hard palate.

Tuberculosis of the salivary glands is very rare. Fioravanti has recently reported one parotid case and the records of thirteen others. Although the writer doubts their being primary in most cases, infection may take place through the blood or lymph streams; also through the ducts of these glands, as it is quite probable that other infections gain their entrance in this

way, the most common of these being epidemic parotitis. However, the fact that the lymph glands in the region of the submaxillary salivary glands are frequently involved, as are also those draining the pharynx and tonsil which are immediately posterior and inferior to the parotid, it can well be imagined that infection may be either direct or hematogenous.

All cases reported show a mixed infection, which may mean that the soil is prepared by previous infection. The writer believes this to be the case, or that possibly a second infection may have become engrafted upon a quiescent tubercle.

The tubercle goes on to caseation, and later to abscess. It may discharge through the duct, although, as a rule, it is necessary to open it. The gland destruction may be partial or complete. In Gilmer's case of sublingual gland it was complete, while in the writer's, a double parotid, it was only partial in each gland.

Pain is usually severe, as the abscesses are walled off from each other by the septa of the gland, and a new abscess forms after one is opened. This may be stopped by opening each pocket, but this is not an easy matter, as, in the case of the parotid, care must be taken not to wound the facial nerve; in case of the submaxillary, the hypoglossal.

The writer's case shows that the nervous tissue is not affected by the process any more than the fibrous tissue septa, for although the pus completely surrounded both nerves, there was only a slight affection of the right nerve, due, the writer believes, to contraction of scar tissue, as symptoms did not appear until after healing had commenced.

Until within the last twenty years lupus was considered as a disease separate and distinct from tuberculosis. Even as late as 1896 there was controversy over the relationship of lupus and tuberculosis, but at present we know that the tubercle bacillus can be found in lupoid tissue.

There seems to be a difference in the mode of infection, or possibly there is a difference in the organism which is not shown either in the tissues or in our present laboratory methods by cultural characteristics. The number of bacilli present is always small. Lupus has little effect on general nutrition and as a rule causes little pain.

Girls, before or about the age of puberty, seem to be most frequently affected. The skin lesions are encountered more

often than those of the mucous membrane, hence these cases usually fall into the hands of the dermatologist.

Hollander suggests that any grade of transition may exist, from the lupoid ulcer with its hypertrophic folds and slow growth, to the ragged tuberculous ulcer, rapidly destroying tissue; and that they may be found side by side in the larynx and in the mouth.

Lupus in the mouth, either primary or secondary, is rare. Langie says: "If secondary lupus is rare, primary lupus is among the rarest of the rare."

Only one case of lupus of the mucous membrane has come under the writer's observation, one involving the nasal mucosa. A number of cases of lupus have been reported in this country. Among them is that of Asch, which was also later reported by E. Mayer. This concerned a patient who had involvement of the soft palate, anterior pillars, tonsil and pharynx.

Interesting also are the two cases of Knight, one a young girl, fourteen years of age, in whom the nose was first attacked and later the lower lip, upper gums, cheek, palate, uvula and nasal septum. The second patient, aged twenty-seven years, showed ulceration of the hard and soft palate and pharynx, with cicatricial bands extending from the tongue to the lateral walls of the pharynx, and from base of tongue to the epiglottis.

Asch's table of ages, although he does not give sex, is also interesting compared with table of ages in tuberculosis of the mouth, and is as follows:

- 4 cases beginning before 10 years of age.
- 6 cases beginning between 10 and 15 years of age.
- 8 cases beginning between 15 and 20 years of age.
- 3 cases beginning between 20 and 30 years of age.
- 1 case beginning between 30 and 40 years of age.
- 1 case beginning at 40 years.

Out of one hundred and sixty-eight cases collected by the writer, in which age and sex are given, there were one hundred and forty-five males and twenty-three females. The age varies from five to eighty years, with an average of thirty-six years.

The diagnosis of primary tuberculosis of the mouth pre-

sents some difficulties. It must first be differentiated from syphilis, which can be done by first differentiating from the primary sore. This is a sharply defined punched-out appearing ulcer, with indurated base, and shows no tubercles. In the case of the secondary manifestations, they are usually multiple; while primary tuberculous lesions, unless of some weeks' standing, would be simple, and the secondaries would have disappeared. The gummata can be differentiated by the lack of tubercles around the edges. Scraping the ulcer, or sectioning the tissue from edge, finding the tubercle or spirochetæ, or at least either the von Pirquet or the Wassermann reactions, will decide.

In differentiating epithelioma from tuberculosis, we observe the indurated edges of epithelioma and the generally soft edges of tuberculous ulcer; the depressed base of epithelioma and presence of many small tubercles in tuberculosis and the age of the patient, of value only when young. Any reaction proving the lesion to be tuberculous is not sufficient, as we may have tubercle engrafted upon epithelioma. Microscopic examination is the only absolute and reliable test.

In secondary disease we may consider against all symptoms in other diseases the history of cough, expectoration, tubercle bacilli in the sputum, loss of weight, hemoptysis, etc., before resorting to other means.

It must be borne in mind, however, that symptoms of pulmonary tuberculosis will not exclude syphilis; and we must not give the therapeutic test for syphilis to patients with pulmonary or any other tuberculous lesion, if it is possible to avoid it, or unless the Wassermann reaction shows that syphilis is also present.

Della Vedova reports twenty-three cases operated upon by Volkman, Bull, and other eminent surgeons under mistaken diagnosis; as was also a case of Shepard. The interference with general health may be found either in cases of epithelioma or tuberculosis.

The first authentic case of lupus of the oral cavity on record was reported by Travers, in 1827, although Louis reported four cases of tuberculosis found at postmortem in 1825; while the first case of ulcer of the tongue was reported by Thomae in a thesis dated 1829, and Rayer in 1835, a case of perforation of palate. However, Schoelermann refers to Fabricius Hil-

danus, who reported a case in 1646 which he believed to be one of oral tuberculosis.

REPORTS OF CASES.

It would be out of place even to attempt to detail the cases reported, the references having multiplied to such an extent in the last few years. Hence, only such cases as show special features of interest have been given prominence; the remainder are shown only in the tables.

Case 1.—Seen at the Jewish Consumptives' Relief Society. Male, student, age twenty-two years. Pulmonary tuberculosis for two years; laryngeal tuberculosis for three months; developed ulceration in the region of the right lower third molar, causing sloughing of the gum and caries of the jaw. Tubercles in tissue surrounding ulcer. Tubercle bacilli found on scraping ulcer.

Case 2.—D. K., physician, thirty-four years of age. Pulmonary tuberculosis for four years; slight laryngeal involvement for six months; developed ulcer on lingual surface of right lower first bicuspid and second bicuspid, first, second and third molars, with ulceration of the lower lip and floor of mouth under tongue. This patient also developed ulceration of the gum in the region of the right upper first molar, which had been absent for several years. Another ulcer in the region of the left upper third molar, which involved the tuberosity. Patient died three months after first involvement of the jaw.

Case 3.—J. F. S., machinist, age thirty-four years. Patient was referred by his dentist, Dr. M. G. Snyder. Tuberculosis for one and a half years; pulmonary, but no laryngeal involvement. After destruction of the pulp of a lower first molar, glands under jaw became swollen, and were opened by the writer three months later. Pus contained tubercle bacilli. Patient developed tuberculosis of the left testicle three and a half years later. At present he is in good health.

Case 4.—H. H. S., male, age thirty years, clerk. Was referred by Dr. C. A. Graham. Gave history of pulmonary involvement for two and a half years; laryngeal involvement for eleven months. First complained of tenderness of tip of tongue, which he attributed to its coming in contact with calculus on lingual surface of anterior inferior teeth. This ap-

peared as a swollen, reddened papilla, which broke down into an ulcer about the size of a pin head. Ulcer continued to enlarge until it reached the size of a navy bean, when the electric cautery was applied to it. Healing took place within a few days. The pulmonary and laryngeal condition progressed, and, although the teeth were kept free from calculus, an ulcer developed in the edge of the scar one year later. This second ulcer was destroyed with the electric cautery. It cicatrized and remained so until death, a few months later.

Case 5.—I. H. E., traveling salesman, aged twenty-eight years. Referred by Dr. G. W. Holden, on account of nasal condition. Pulmonary tuberculosis, two years. One year later he complained of irritation around roots of broken down upper right first molar, which continued to spread along the exterior, involving the gums around second and third molars; later also involved the palatal surface. Scrapings showed tubercle bacilli. Teeth were extracted; ulcer cauterized with electric cautery, and although this was repeated several times, it was without success. Ulcer was kept within these limits for about three years, when patient died of brain embolus.

Case 6.—A. M. B., cigar maker, age twenty-nine years. Referred by Dr. F. A. Greedy on account of ulceration of alveolus and gums between upper left central incisor and cuspid, lateral being absent, and about five centimeters from gum margin. Ulcerated surface showed tubercles, and tubercles were also seen in surrounding tissues. This patient also had involvement of the left maxillary sinus, which may possibly have been tuberculous. He had been a sufferer from pulmonary tuberculosis for eight years. The ulcer healed after numerous cauterizations with trichloracetic acid, and remained so until death from pulmonary tuberculosis, two years later.

Case 7.—G. R., barber, age thirty-nine years. Consulted writer for laryngeal tuberculosis. Pulmonary tuberculosis, one and a half years; laryngeal involvement, about one month, the latter showing rapid progress, for at the time of examination, vocal cords, ventricular bands, interarytenoid sulcus, and epiglottis, all showed marked infiltration. One month later patient noticed what he called a blister on the dorsum of the tongue, and, when called to my attention, showed a small, slit-like opening in center, with ulcerated base, showing overhang-

ing edges. Ulcer showed no tendency to heal and no tendency to spread. Patient died six weeks later.

Case 8.—S. R., merchant, age thirty-nine years. Was referred by his dentist, Dr. M. H. Barrin, on account of involvement of socket of the left lower first bicuspid. Pulmonary involvement for five years. On examination pus was found oozing from the socket, with ulceration showing tubercles of the surrounding gums. Scraping showed tubercle bacilli. Patient was lost sight of after first treatment.

Case 9.—J. R., tailor, age thirty-four years. Was referred by Dr. Philip Hillkowitz, on account of ulceration of the right border and inferior surface of the tongue. Pulmonary involvement of four years' standing. Ulcer cauterized with electric cautery several times with no appreciable benefit, and it continued to spread. Patient died of tuberculosis three months after ulcer was first seen.

Case 10.—N. H., male, nurse, age forty years. Referred by Dr. Fish, from the Jewish Consumptives' Relief Society, on account of involvement of the left side of the lower jaw in the region of the second bicuspid. Pulmonary involvement for three years. Gum ulcerated, and necrosis with the formation of small sequestrum. Removed by operation, parts cauterized with the electric cautery. This case had been operated previously, but disease continued to spread. Tubercle bacilli found in material curetted from ulcer. Patient alive and in good health.

Case 11.—Patient from Jewish Consumptives' Relief Society, male, aged forty years. Referred by Dr. C. D. Spivak. Pulmonary involvement for two years. Fissured ulcer of tip of tongue, cauterized with trichloracetic acid. This patient was seen by the author only three times, so that final result cannot be recorded.

Case 12.—H. A., patient at National Jewish Hospital for Consumptives, age twenty-four years. Pulmonary involvement of two years' duration. Showed swelling of left parotid gland, which later suppurated, and on opening pus was found to contain only the diplococci resembling those found in epidemic parotitis. A second pocket of pus, opened a few days later, showed tubercle bacilli as well as diplococci. The left gland suppurated five weeks later, and on opening, showed mixed infection, tubercle bacilli and diplococci. Case progressed

slowly, each side showing several pockets of pus; right had entirely healed when patient was last seen, and gland still showed some function, although the amount of salivary secretion was small. Left side still showed some secretion when patient was last heard from.

Case 13.—Young man, age twenty years. Seen on my service at the Denver City and County Hospital. Developed an abscess of the right upper first molar, which was manifested by a large swelling of the cheek, and a discharge of pus through Stenson's duct. Apparently abscess had eroded wall of duct. Pus contained numerous caseous masses, and patient was suffering from pulmonary tuberculosis of several months' standing.

Case 14.—Miss R., school teacher, age forty-two years. Referred by Dr. J. R. Arneill, on account of her laryngeal condition. Larynx showed infiltration of vocal cords, ventricular bands, and arytenoepiglottidean folds, with ulceration of the interarytenoid sulcus and left ventricular band. Three weeks later the patient called attention to a small "blister" upon the dorsum of the tongue, which, on examination, showed a fissured opening in the center. The condition in this case was very similar to that in case 7. Patient died one week after ulcer appeared.

Case 15.—Mr. C. B., age twenty-two years. Referred by Dr. J. H. McKay, on account of laryngeal tuberculosis. Patient was also suffering from acute pulmonary tuberculosis; history of syphilitic infection two years previous. On examination larynx showed infiltration and ulceration of entire surface. There was also ulceration of the left side of the pharynx, posterior pillar and tonsil; palate later became involved. Death six weeks after first visit.

Case 16.—D. S. R., age twenty-six years. Referred by Dr. L. H. Schultz. Pulmonary tuberculosis, one and a half years; laryngeal tuberculosis, four months. Gums upon the left upper jaw, on both palatal and buccal surfaces, are ulcerated from the second bicuspid to and including the tuberosity. On the right upper jaw, ulceration is found on the gum on the buccal surface of the first molar; another ulcer is found on the lingual surface of the lower jaw on the left side opposite first molar. Tubercle bacilli found on scraping.

Case 17.—Japanese, age thirty-six years. Seen in consul-

tation with Dr. A. L. Bennett. On examination a tumor-like mass was found in the middle of the dorsum of the tongue the size of a hickory nut, which was quite hard, but showed slight evidence of fluctuation. Upon opening about a dram of caseous pus was evacuated, which was not examined. Abscess healed without complication. No evidence of tuberculosis in this patient, but brother had died of tuberculosis.

The following tables show 534 cases with 661 lesions, distributed as follows:

Tonsil	48
Pharynx	51
Palate	97
Upper jaw	48
Lower jaw	36
Cheeks	16
Tongue	277
Lips	48
Location not specified.....	40

No.	Author.	Cases	Date	Lips	Cheek	U. Jaw	L. Jaw	Tongue	Tonsil	Pharynx	Palate	Mouth	PUBLICATION.
1	Delevan (personal)....	*P. 3	1887	1P	1P 1S	1S	1S	1P	22	New York Med. Jour., XLV, 536-541.
2	From other sources....	*S. 4	1P 1S	1S	1S	1P	Med. News, Philadelphia, May 29, 1886.
3	Stewart,?	1886	1	Jour. Amer. Med. Asso., August 12.
4	Theisen, C. F.	1889	Brit. Med. Jour., 1887, II, 456.
5	Lubinski,?	1887	New York Med. Jour., XI, 505-507.
6	De Blois,?	1884	Rev. de Laryngologie, No. 20.
7	Ragoneau,?	1891	France Med., Paris, XXVI, 113.
8	Routier, A.	1878	1	1	Jour. Laryn. and Rhin., January.
9	Battle,?	1878	Cyclopaedia, VI, 986.
10	Von Zeimssen,?	1890	Diseases of Tongue.
11	Butlin and Spencer,?	1900	1	Jour. Laryn. and Rhin., April 5.
12	Machtyre,?	1891	Societe Med. des Hop., November 29.
13	Barth,?	1880	Quoted by Baudon.
14	Bucquoy,?	1887	1	Quoted by De Brun, Du Bois Noir.
15	Gosselin and Fano,?	Cong. Internat. de Laryngol. 1880, Milan, 1882, I.
16	Lennox Browne,?	1881	1	104-112.
17	Kronhelm,?	Langenbeck's Schrn. Archiv f. Klin. Chir., Suppl., Band 2, XXI.
18	Bosworth,?	Trans. Amer. Laryn. Asso., 1881, III, 65-77.
19	Duplay,?	1883	Quoted by Euteneuer.
20	Fournier,?	Quoted by Euteneuer.

*P. Primary. S. Secondary.

No.	Author.	Cases	Date	Lips	Cheek	U. Jaw	L. Jaw	Tongue	Tonsil	Pharynx	Palate	Mouth	PUBLICATION.
20	Uchermann	*P. 3.	1884	1	Norsk Mag. f. Laegendersk, Kerstiania, XIV.
21	Abercrombie and Gay. 3	1886	3	3	Brit. Med. Jour., London, II, 923.
22	Kidd, Percy 11	1886	7	4	Discussion No. 21, Ibid.
23	Feurer, G. 2	1886	1	3	Switzerland Correspondence, Brit. Med. Jour., II.
24	Chamberlin, C. W. 2	1881	1	1	Proc. Conn. State Med. Soc.
25	Abrahams, P. S. 1	1885	1	1	Trans. Acad. Med., Ireland, Dublin, III, 358.
26	Stchastny, A. 5	1889	5	Annals de l'Inst. Pasteur, Paris, III, 224.
27	Stonham	1889	1	Westminster Hospital.
28	Wingrave, V. H. W. 1	1892	1	Brit. Med. Jour., II.
29	Ducrot, L. 2	1879	1	2	Etude sur la tuberculose de la bouche, etc.
30	Browne, L., and Grant, Dundas	1881	1	1	1	Archives of Laryngology, January.
31	Babes 1	1883 1	Bull. Soc. Anat. de Paris, LVIII, 341.
32	Elchoff, J. 1	1881 1	1	1H	Deutsche med. Woch., Berlin, VIII, 418.
33	Boeckel, E.	1872	1	Gaz. med. de Strasb., XXXII, 8.
34	Finger, E. 3	1883 2	1	1	1	1	Med. chir. Centralb., Wien, XX, 447.
35	Hanseman, D.	1885	Arch. f. path. Anat., etc., Berlin, CHH.
36	Grocler 4	1886 3 2	1	1H	Bull. de Laryngol., etc., March, 30.
37	Jullard fils. 2	1901	1	2	2	8	Bull. Soc. Med. de la Suisse Rom., Lausanne, IV, 104.
38	Le Dentu, A. 1	1875 1 1	1	1	Assoc. franc. jour. l'arance d. sc. Compt. rend. 187
	 1	1876 1	1	1	b. Paris, 1877, V, 884.

No.	Author.	Cases	Date	Lips	Cheek	U. Jaw	L. Jaw	Tongue	Tonsil	Pharynx	Palate	Mouth	PUBLICATION.
63	De Brun, Du Bois, Noir.	*P. 1	1882	1	Bull. Soc. Clin. de Paris, VI, 276.
64	Schott	1883	1	Quoted by Chvostek.
65	Charlier	1891	1	J. d. Mal. Cutan. et Syph., Paris, III.
66	Chvostek	1884	1	Algem. Wien. med. Ztg., XXIX, 209-224, 234-60-89, 79.
67	Dowd, C. N.	1890	1	Proc. New York Path. Soc., 1890-91.
68	Dugnet	1885	1	Ann. Med. Chir. franc. et etrang. Paris, I, 185-495.
69	Fercot	1872	1	Union Med. Paris, 3s. XIV, 716-29.
70	Fox, S. A.	1889	1	1	Gaillard's M. J., New York, XLVII, 518.
71	Gade, F. G.	1884	1	Norsk Mag. f. Laegerindensk, Christiania, XIV, 95.
72	Godlee, R. J.	1883	3	Trans. Path. Soc., London, XXXV, 184.
73	Hadden, W. B.	1882	1	Trans. Path. Soc., London, XXXIV, 135.
74	Heurtaux	1882	1	Bull. Soc. Anat. de Nantes, Paris, VI, 101.
75	Jackson, V.	1886	1 tip	Lancet, London, II, 444.
76	Jessett, F. B.	1883	1	Trans. Path. Soc., London, XXXV, 162.
77	König, C. F. W.	1857	1	Org. f. d. ges. Heilk., Berlin, VI, 101.
78	Korte, W.	1876	1	Deutsche Ztschr. f. Chir., VI, 442.
79	Kuessler, B.	1876	2	Deutsche med. Woch., VII, 277.
80	Laboulbene, H.	1874	1	Courier Med., Paris, XXIV, 323.
81	Lancereux, E.	1874	1	Bull. Soc. Med. d. Hop. de Paris, 2s. 8, 298.
82	Laurent	1884	1	Lyons Med., XIV, 513.
83	Leger, H.	1887	1	Ann. de Mal. de l'oreille, du larynx, etc., Paris, III, 35.

No.	Author.	Cases	Date	Lips	Cheek	U. Jaw	L. Jaw	Tongue	Tonsil	Pharynx	Palate	Mouth	PUBLICATION.
111	Albrecht	ap. 1	1891	1	1	1	1	1	1	1	1	1	Pathologie und Therapie der Lungenschwindsucht, Wien, 1908.
112	Weber, O.	1	1891	1	1	1	1	1	1	1	1	1	Pitha, Billroth Chirurgie III, I.
113	Bamberger	7	1891	1	1	1	1	1	1	1	1	1	Virchow's Spec. Path. u Ther., Ed. 6, I.
114	Barling, C.	2	1889	1	1	1	1	1	1	1	1	1	Lancet, I, 1423.
115	Bean, C. E.	2	1889	1	1	1	1	1	1	1	1	1	Trans. Am. Laryn. Asso., XI, 113-16.
116	Burnheim, S.	1	1900	1	1	1	1	1	1	1	1	1	Dental Cosmos, 1901, Vol. XLIII.
117	Paget, James.	1	1858	1	1	1	1	1	1	1	1	1	Med. Times and Gaz.
118	Fleming	1	1850	1	1	1	1	1	1	1	1	1	Dublin Quarterly Jour., August, No. XIV.
119	Moeller	1	1910	1	1	1	1	1	1	1	1	1	Muench. med. Woch., No. 2.
120	Terenkoczy	1	1889	1	1	1	1	1	1	1	1	1	Abst. Jour. Laryn., Rhin. and Otol., July, 1889.
121	Valude	1	1888	1	1	1	1	1	1	1	1	1	Abst. Jour. Laryn., Rhin. and Otol., London, December.
122	Gleitsman	1	1888	1	1	1	1	1	1	1	1	1	Academy of Med., November 27.
123	Dunn, J.	1	1904	1	1	1	1	1	1	1	1	1	Virginia Med. Semi-Monthly, Richmond, IX, 486.
124	Jaruntovsky	1	1895	1	1	1	1	1	1	1	1	1	Abst. Jour. Laryn., Rhin. and Otol., July.
125	Semont, Felix	1	1895	1	1	1	1	1	1	1	1	1	Jour. Laryn., Rhin. and Otol., December.
126	Cameron, H.	2	1896	1	1	1	1	1	1	1	1	1	Glasgow Med. Jour., August, 96.
127	Harland, W. G. B.	2	1903	1	1	1	1	1	1	1	1	1	Proceedings Phila. Co. Med. Soc., n. s., V, 192.
128	Blumenfeld	1	1898	1	1	1	1	1	1	1	1	1	Quoted by Lenzmann.
129	Lenzmann	1	1898	1	1	1	1	1	1	1	1	1	Münchener med. Woch., June 21.

130	Horne, Jobson	1	1897	1	1	1	1	1	1	1	Laryn. Soc. of London. Reported in Jour. Laryn. Rhin. and Otol.
131	Paget, Stephen	1	1890	1	1	1	1	1	1	1	Abs. from Lancet, March 22, 1890, in Jour. of Laryn. and Rhin.
132	Michelson, P.	2	1890	2	2	2	2	2	2	2	Ztschr. f. klin. Med., Berlin, XVII.
133	Lord	1	1893	1	1	1	1	1	1	1	Deut. med. Woch., XV, 1075.
134	Schuchardt	1	1889	1	1	1	1	1	1	1	Quoted by Armstrong.
135	Volkmann	2	1890	2	2	2	2	2	2	2	Annals of Surg., April.
136	Armstrong, G. E.	1	1910	1	1	1	1	1	1	1	Wien. med. Presse., 721.
137	Rethi, L.	1	1893	1	1	1	1	1	1	1	4 Recherches sur la Phthisie, Paris, Bosworth.
138	Louis	2	1825	2	2	2	2	2	2	2	Prager Vierteljahrsschrift, XIII, Part II, 10.
139	Willigk	2	1856	2	2	2	2	2	2	2	Ann. de dermat et syph., Paris, 3s, VII, 286.
140	Thibierge, G.	1	1897	1	1	1	1	1	1	1	These de Paris, 1872.
141	Gazagne	1	1872	1	1	1	1	1	1	1	These de Paris, 1872.
142	Buzenet	1	1858	1	1	1	1	1	1	1	These de Paris. Referred to by Ducrot.
143	Laveran	1	1858	1	1	1	1	1	1	1	1 Referred to by Hansemann.
144	Rocord	6	1890	6	6	6	6	6	6	6	Referred to by Hansemann.
145	Frankel, A.	1	1890	1	1	1	1	1	1	1	Referred to by Hansemann.
146	Langie	1	1890	1	1	1	1	1	1	1	1 Przegia Lekarski keatow. Quoted by Mayer.
147	Travers	1	1829	1	1	1	1	1	1	1	Quoted by Langie, who was quoted by E. Mayer.
148	Mayer, E.	1	1897	1	1	1	1	1	1	1	Trans. Am. Laryn. Assn.
149	Wylie, A., and Win- grave, W.	1	1906	1	1	1	1	1	1	1	Med. Press. and Circular, London, N. S., LXXXII, 639.
150	Billroth	2	1890	2	2	2	2	2	2	2	Quoted by Bowlby.
151	Campbell, J. T.	1	1907	1	1	1	1	1	1	1	Laryngoscope, XVII, 233.
152	Della Vedova	1	1907	1	1	1	1	1	1	1	Policlin, Roma, XIV, Sez. Chir.
153	Tixer and Barjon	1	1907	1	1	1	1	1	1	1	Lyon Med., CVIII, 782.
154	Augier	1	1907	1	1	1	1	1	1	1	Jour. d. scien. med. de Lille, I, 546.
155	Richardson	1	1898	1	1	1	1	1	1	1	Trans. Am. Med. Asso., Vol. I, 479.
156	Cook, George W.	5	1899	5	5	5	5	5	5	5	Dental Review, February, 1899.

*P. Primary. S. Secondary. G. Gums.

†Collected from literature. Operated on mistaken diagnosis.

No.	Author.	Cases	Date	Lips	Cheek	U. Jaw	L. Jaw	Tongue	Tonsil	Pharynx	Palate	Mouth	PUBLICATION.
157	Webber, A.	3P. 2S.	1907	1	2	München. med. Wochenschr., LIV, 1785.
158	Knight	3	1881	1	1	1	2	Trans. Am. Laryn.
159	Hollander	1906	12	30	1	Berl. klin. Woch., June 4.
160	Levy, Robert	8	1908	2	1	1	3	1	Denver Medical Times, XXVII, 197.
161	Sharp, J. C.	1908	1 tip	1	1	1	Amer. Med., Burlington, 385.
162	Lockard, L. B.	14	1909	1	1	1	2	7	2	12	Tuberculosis of Nose and Throat; also personal communications.
163	Grünwald	1	3	1	1H 1S	Atlas Mouth, Pharynx and Nose.
164	Leshure, John	1	2	Abstract.
165	Dally, J. F. H.	1903	1	1	Brit. Med. Jour., p. 1463.
166	Harsha, W. M.	1	1908	1	Surg. Gyn. and Obs., Chicago, VI, 287.
167	Provera, C.	1906	1	Morgagnis, Milano, XLVIII, 27.
168	Guinon, L., and Barbier	1	1908	1	Bull. Soc. de pediat. de Paris, X, 197.
169	Walsham	3	1905	2	1	Channels of Infection in Tuberculosis.
170	Lack, L.	1903	1	Jour. of Laryn., Rhin. and Otol., April.
171	Bezold and Gideonsen	1907	1	Path. Ther. der Kehlkopf Tuberk., Berlin.
172	Rayer	Traite des maladies de la Peau.
173	Rayer	1835	1H	Archives of Laryn., II, No. 2.
174	Cohen	1	1881	1	Dental Review, XXIII, 491.
175	Gilmer, T. L.	4	1909	1	2	1	Referred to by Asch.
176	Paul	1872	These de Paris, No. 37.
177	Fougere, M.	1871	1	1H-S
178	Isambert	1871	1	Memoirs, 107.

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200	Ravenel.....	Amer. Jour. Med. Sciences, Vol. 134, p. 469, 1907.
201	Fabricius Hildanus.....	Opera Cent. III, observat LXXXIV, 1646. Quoted by Scholerman.
202	Wood, George B.....	Laryngoscope, May, 1906.
203	Robertson.....	Jour. A. M. A., November 24, 1906.
204	Goodale.....	Laryngoscope, December, 1906.
205	Lake.....	Laryngeal phthisis.
206	Frankel, E.....	Virch. Arch., CXXI, p. 523.
207	Orth.....	Lehr. der spec. path. Anat., p. 320.
208	Adami and Nichols.....	Systemic Pathology, II, p. 308, 1909.
209	McFarland.....	Pathology, 1910, p. 470.
210	Ribbert.....	Deut. med. Woch., 1902, No. 17.
211	Aubrecht.....	Pathologie und Therapie der Lungenschwindsucht, Wien, 1908.
212	Baumgarten.....	Wiener med. Woch., November 2, 1901.
213	Cornet.....	Volume on Tuberculosis, Nothnagel's Encyclopedia.
214	Bordnave.....	1878, Paris, 61, p. 40, No. 424.
215	Gaucher.....	Prog. Med., 1879, VII.
216	Zaerthial.....	Wiener med. Presse, 1880, XXI. Quoted by Bosworth.
217	Gougenheim.....	Union Med., Paris, 1812, 3rd Series, XXXIV, 1883.
218	Schnitzler.....	1883.
219	Cadier.....	1883.
220	Strassman, F.....	Virchow's Archives, Band. LXIX, Heft 2, Arch. f. path. Anat., etc., Berlin, 1884.
221	Desplous, J.....	De la tuber. de l'arrièrbonde.
222	Gelade.....	De la tuberculose bucco-pharynges.
223	Peter.....	Gaz. Med. de Paris, 63, II, 6, 18, 1880.
224	Testi, A.....	Boll. d. hosp. di Ferno I.
225	Barth, H.....	Bull. et Mem. Soc. d. Hop. de Paris, 3s, IV, 1887.
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230 Pavlicki.....
231 Remy, C.....
232 Rochelt, E.....
233 Thomayer, J.....
234 Tolson, J.....
235 Tolland, H. H.....
236 Trelat.....
237 White, W. H.....
238 Wigelsworth, J.....
239 Zugmondy.....
240 Schliepegrell, J.....
241 Schlsferowitsch, P.....

242 Tarrade, F.....

243 Zandy, C.....
244 Brindel.....
245 Campbell, C.....
246 Combemale, F.....
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248 Derville, L.....
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250 Garel and Armand.....
251 Gaston and Chompret.....
252 Greve, C.....
253 Keen, W. W.....
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255	Krakht. S. F.	Protok. Mosk. Ven. i dermat. Obsh. VII, 1897-1898.
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257	Morestin, H.	Bull. et Mem. Soc. Anat. de Paris, LXXIX, 1904.
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FIGURE I.

Case 2. Tuberculosis of lower jaw. The superior maxilla was affected on both sides a few weeks before death.*

*The illustrations are all taken from cases of the author, although Figures 1, 2, 8, 9, 13, 14, and 15 were loaned to Dr. L. B. Lockard and published in his book, "Tuberculosis of the Nose and Throat."

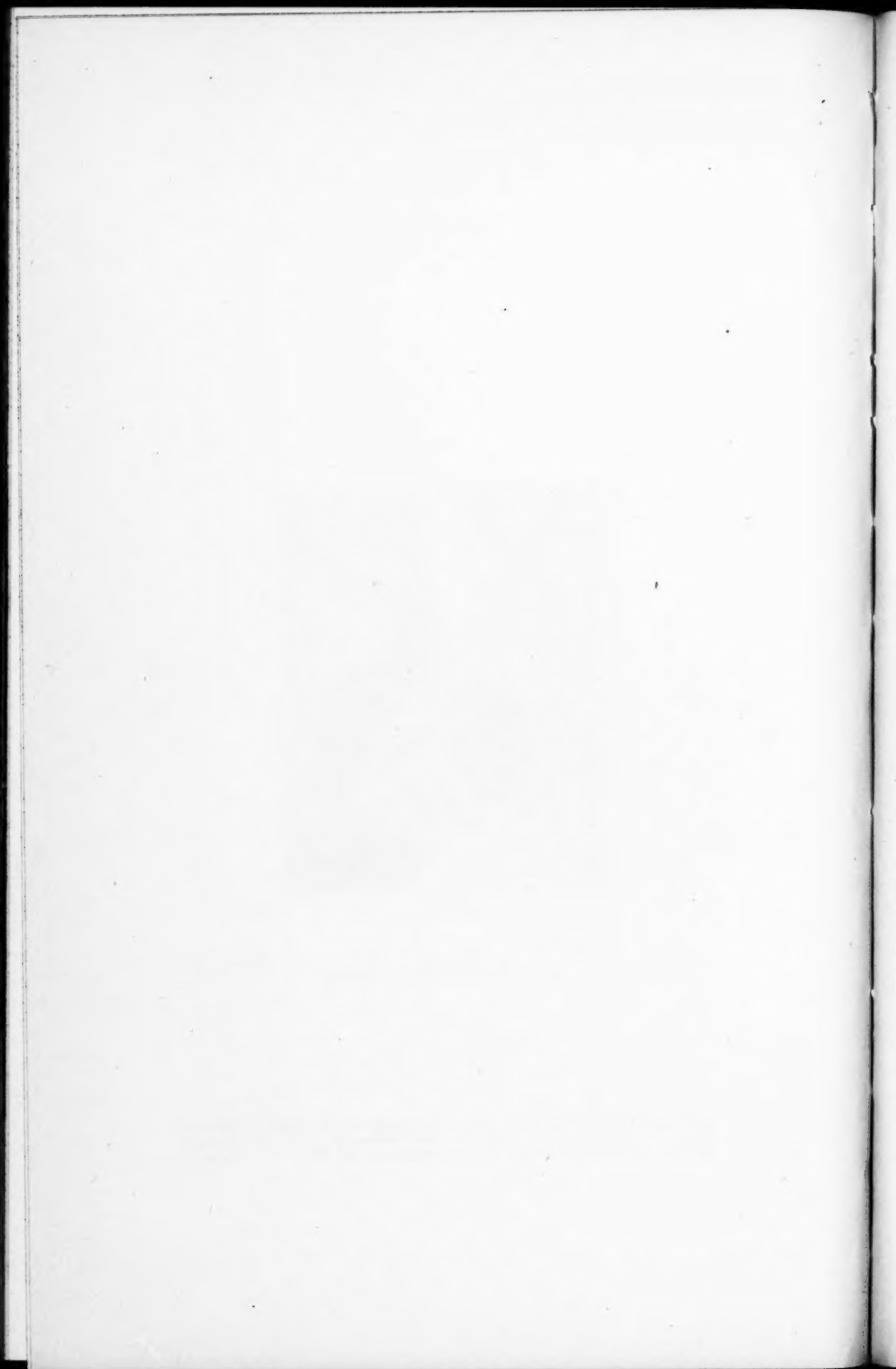




FIGURE II.

Case 6. Tuberculous ulcer following abscess of left superior lateral incisor.

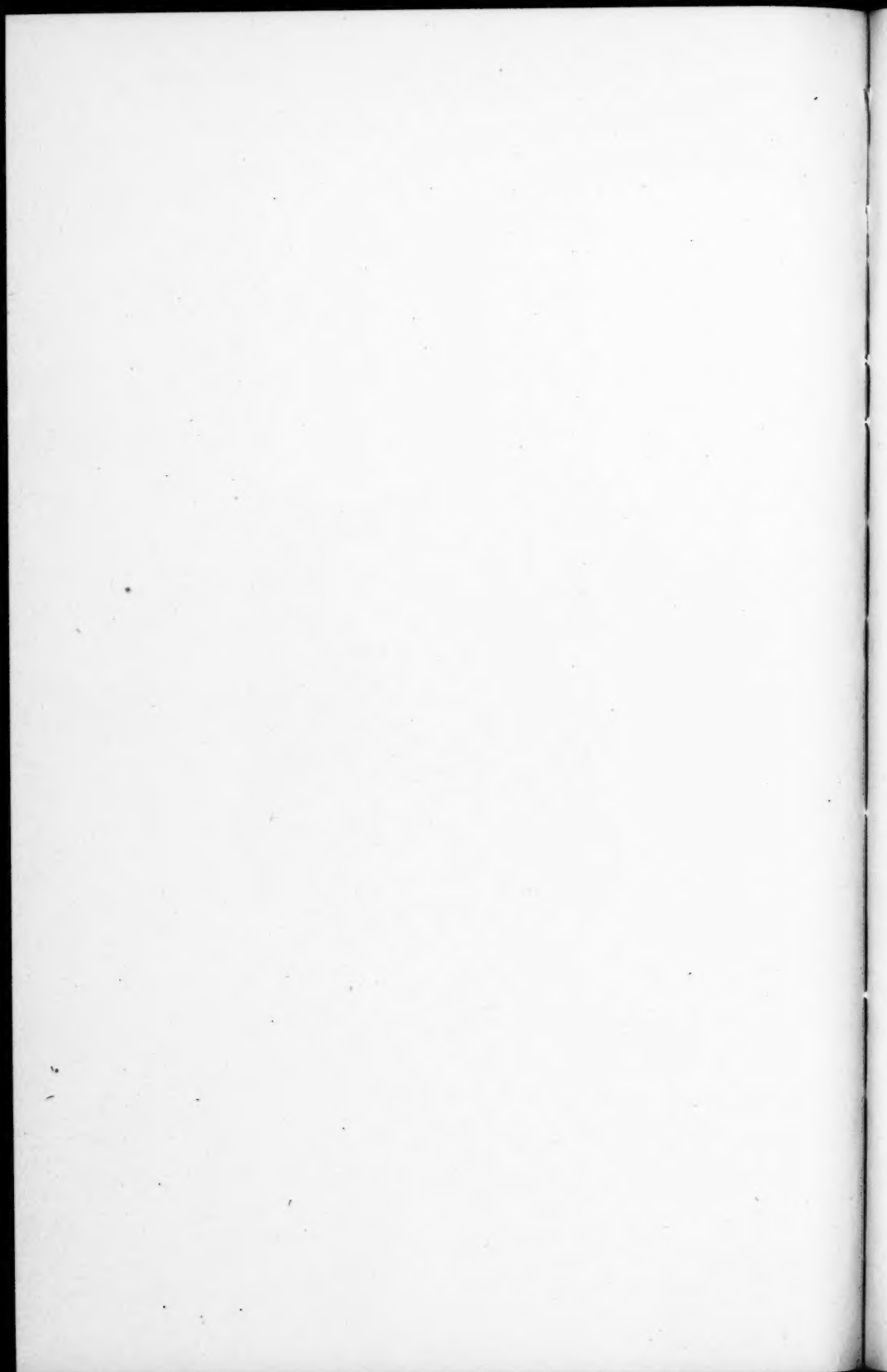




FIGURE III.

Case 3. Submaxillary lymphatics infected from lower first molar.





FIGURE IV.

Case 3. Same patient three and one-half years later.

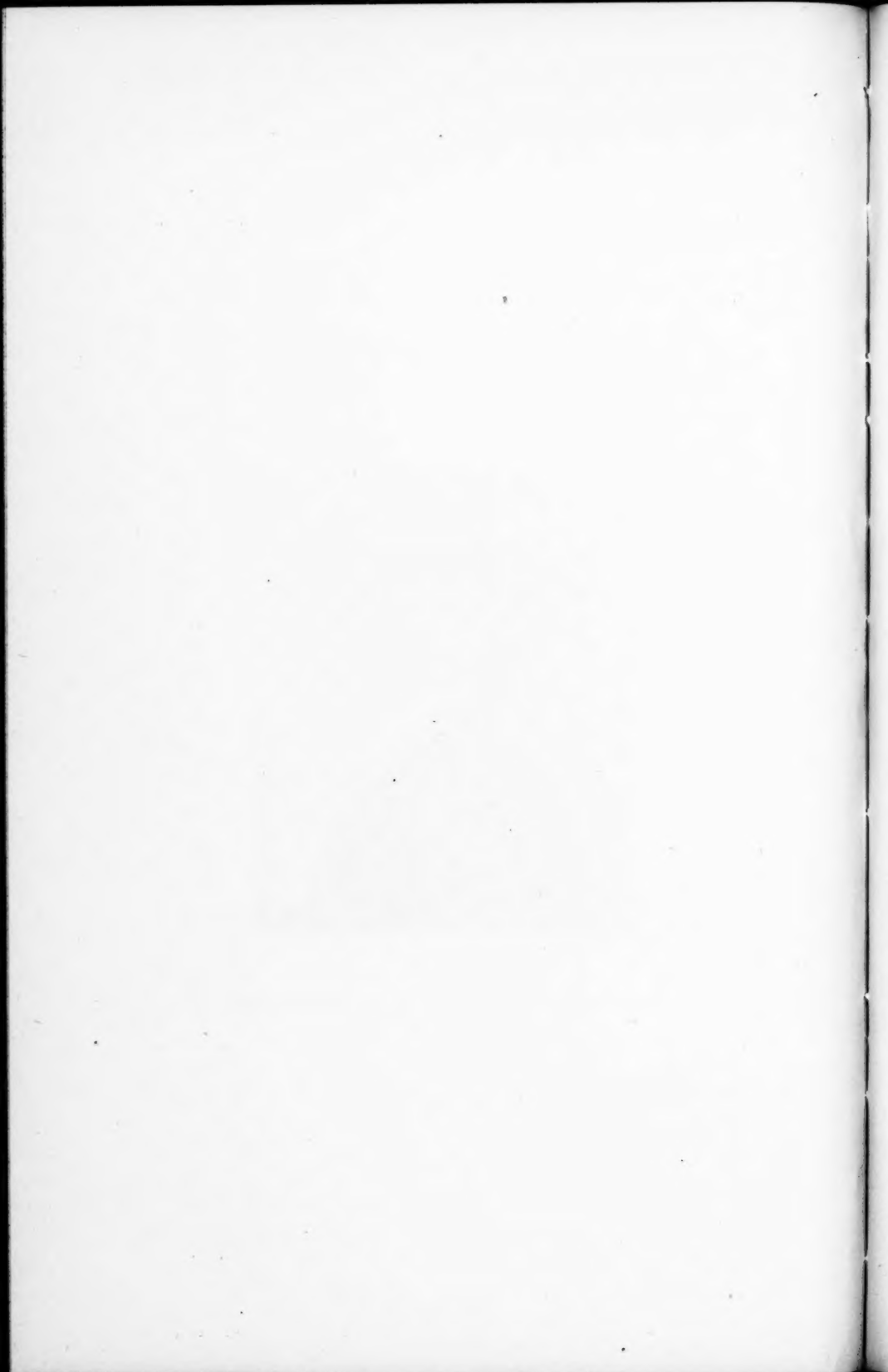




FIGURE V.

Case 4. Tuberculous ulcer of tongue.



FIGURE VI.

Case 12. Tuberculosis of left parotid gland.

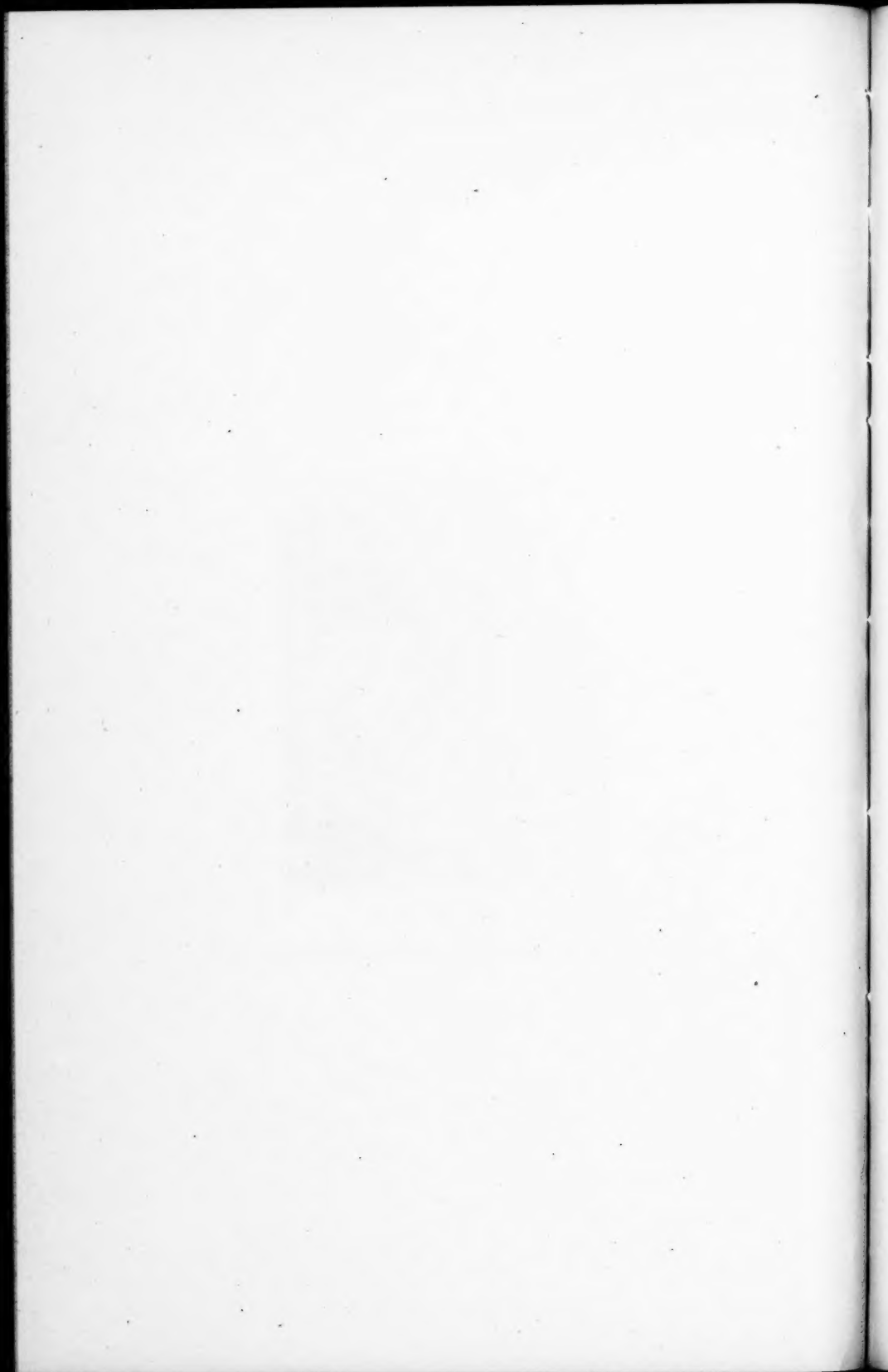




FIGURE VII.

Case 12. Tuberculosis of right parotid gland.

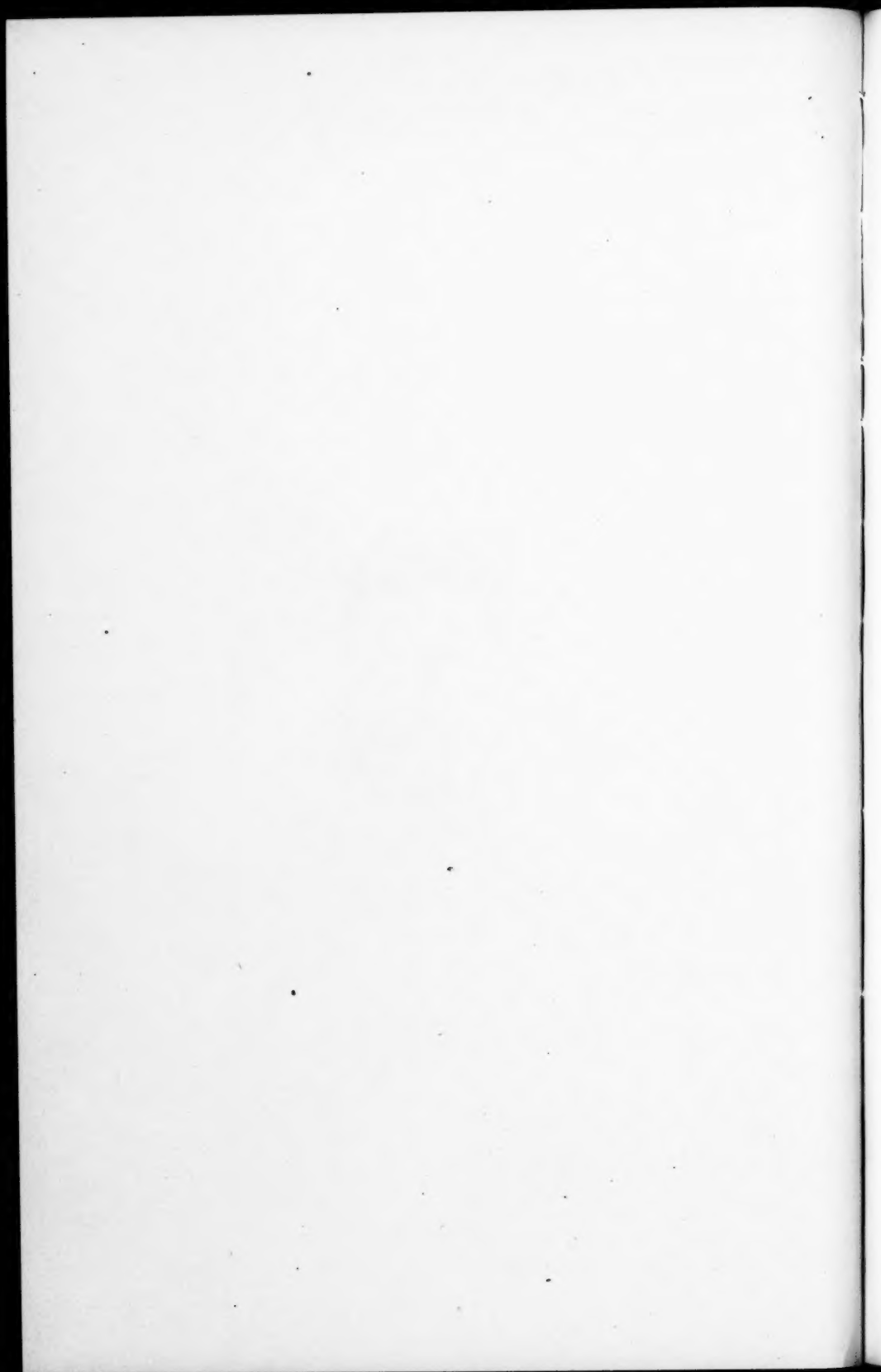




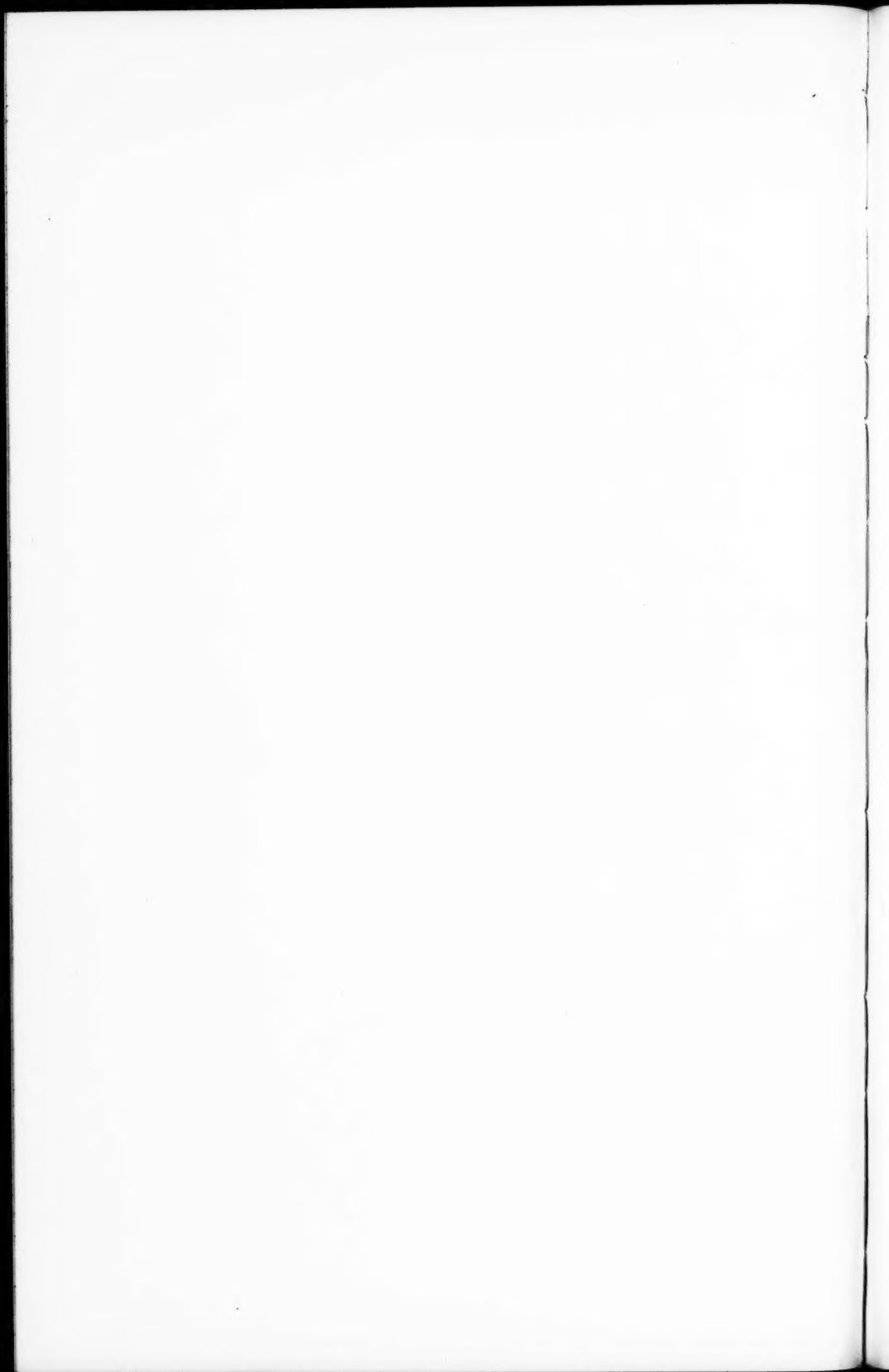
FIGURE VIII.

Case 4. Tuberculosis of tongue. Patient, male, thirty-one years of age.



FIGURE IX.

Case 7. Lesion similar to case 14. Tuberculosis of tongue resembling gumma. Patient, male, thirty-four years of age.



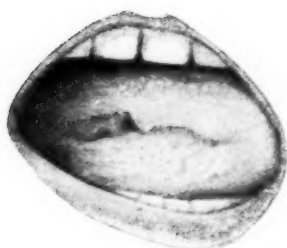


FIGURE X. *Cavallo*

Case 9. Tuberculosis of border and under surface of tongue.



FIGURE XI.

Case 11. Fissured ulcer of tip of tongue.

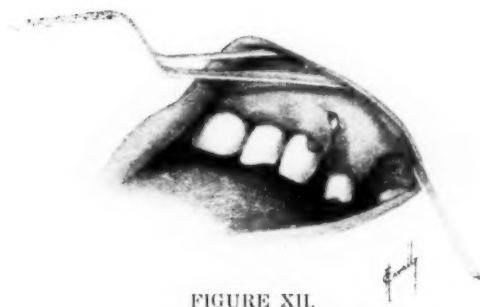


FIGURE XII.

Case 4. Tuberculosis of alveolus of left superior maxilla.

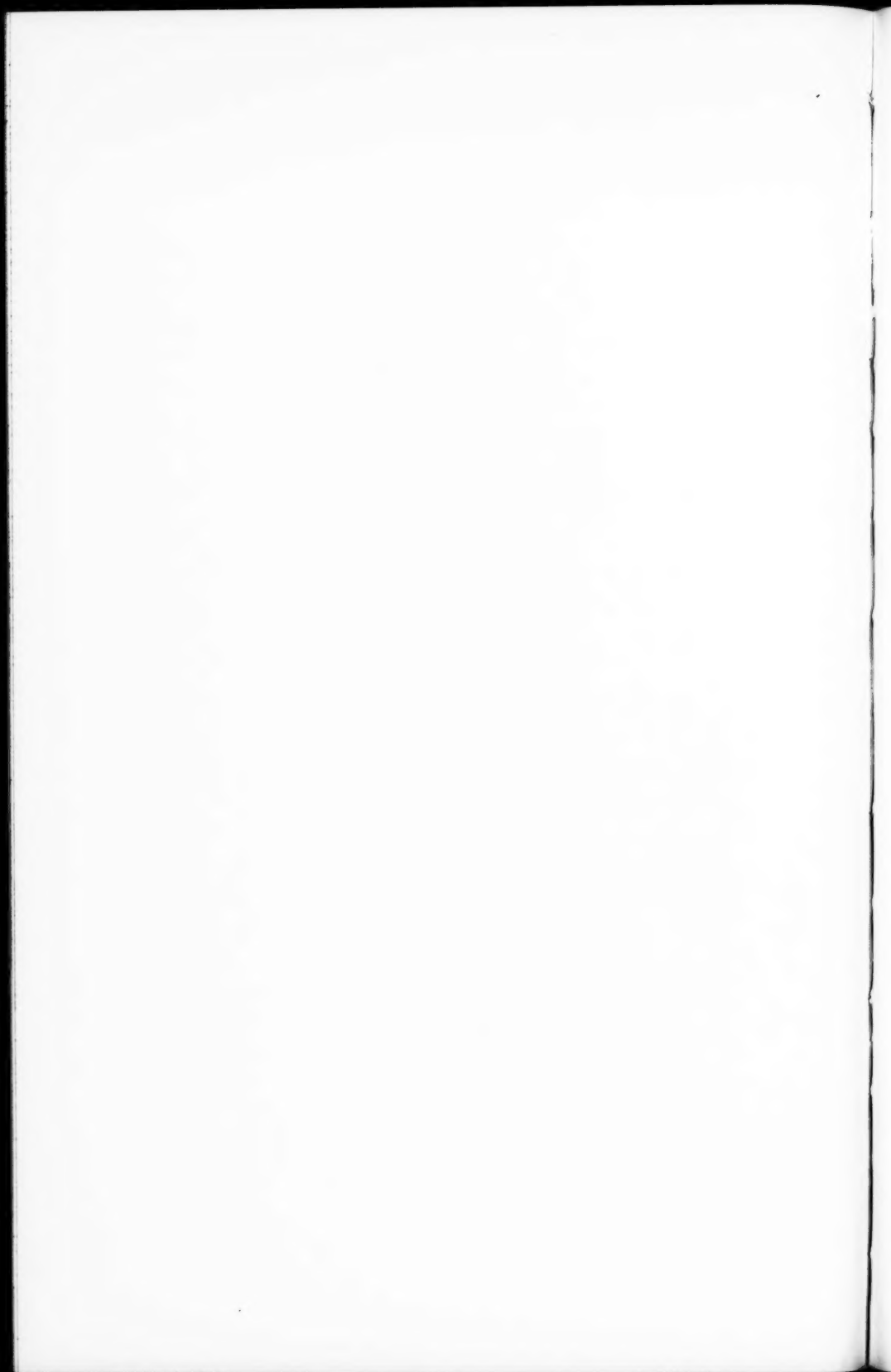




FIGURE XIII.

Specimen from case 2. Tuberculosis of mandible and lower lip.
Male, thirty-four years of age.



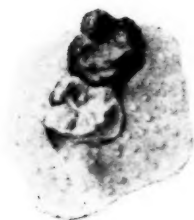


FIGURE XIV.

Case 5. Tuberculosis of tuberosity of upper jaw. Patient, male, twenty-seven years of age.

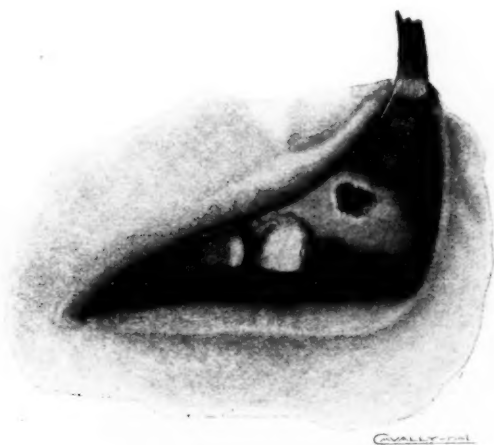
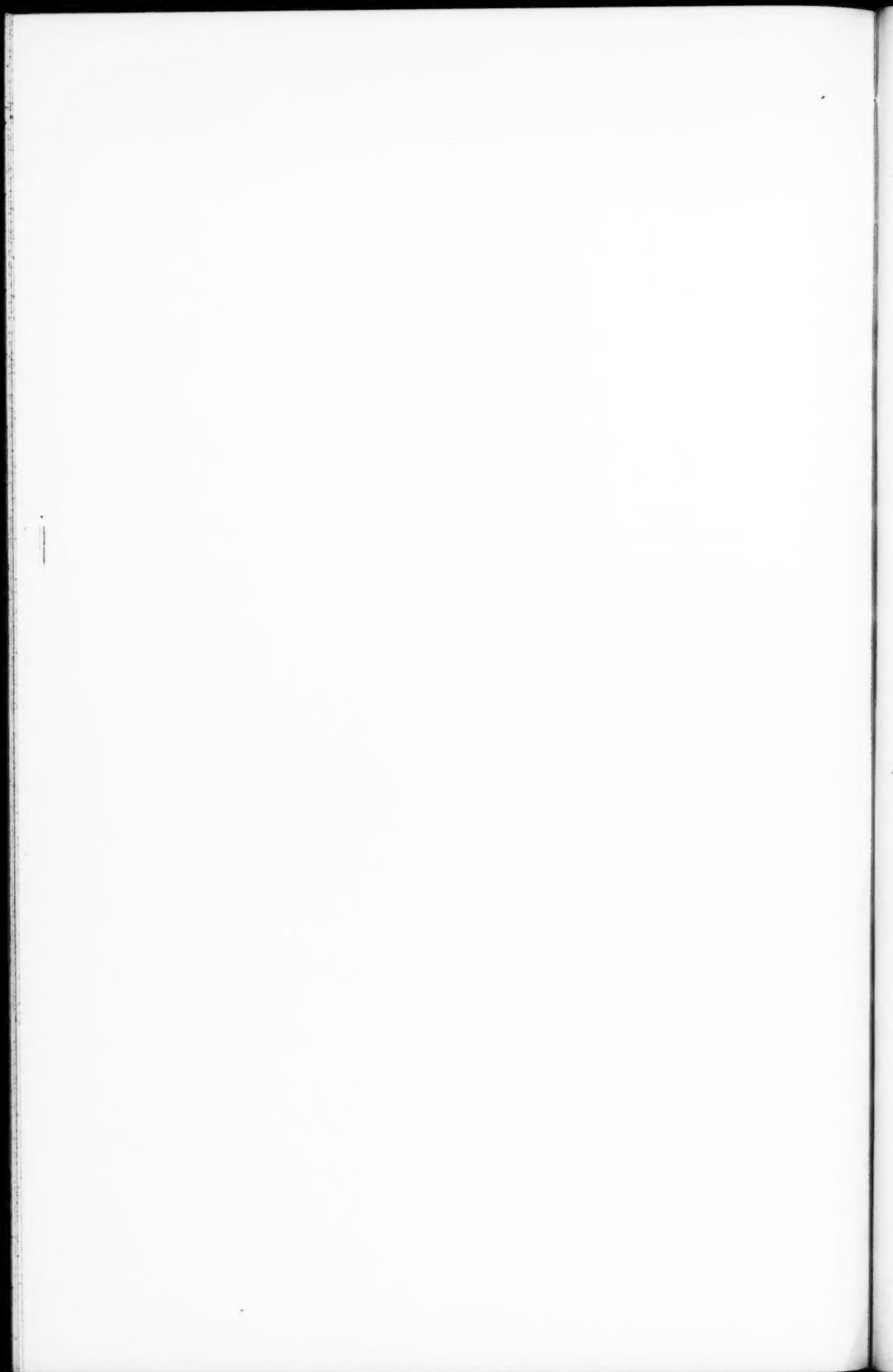


FIGURE XV.

Case 6. Tuberculosis of upper jaw. Patient, male, twenty-nine years of age.



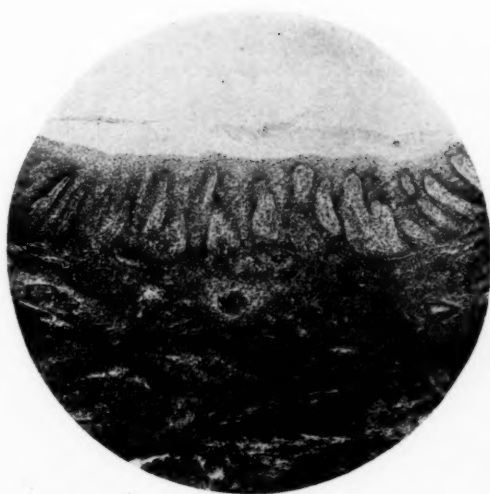


FIGURE XVI.

Case 16. Showing relation of tubercle to epithelium. Tissue taken from gum margin between first and second left lower molars.





FIGURE XVII.

Case 4. Showing tubercle under epithelium and a very much degenerated tubercle in deeper tissues. Same as Figure 16.





FIGURE XVIII.

Showing partially broken down tubercle.

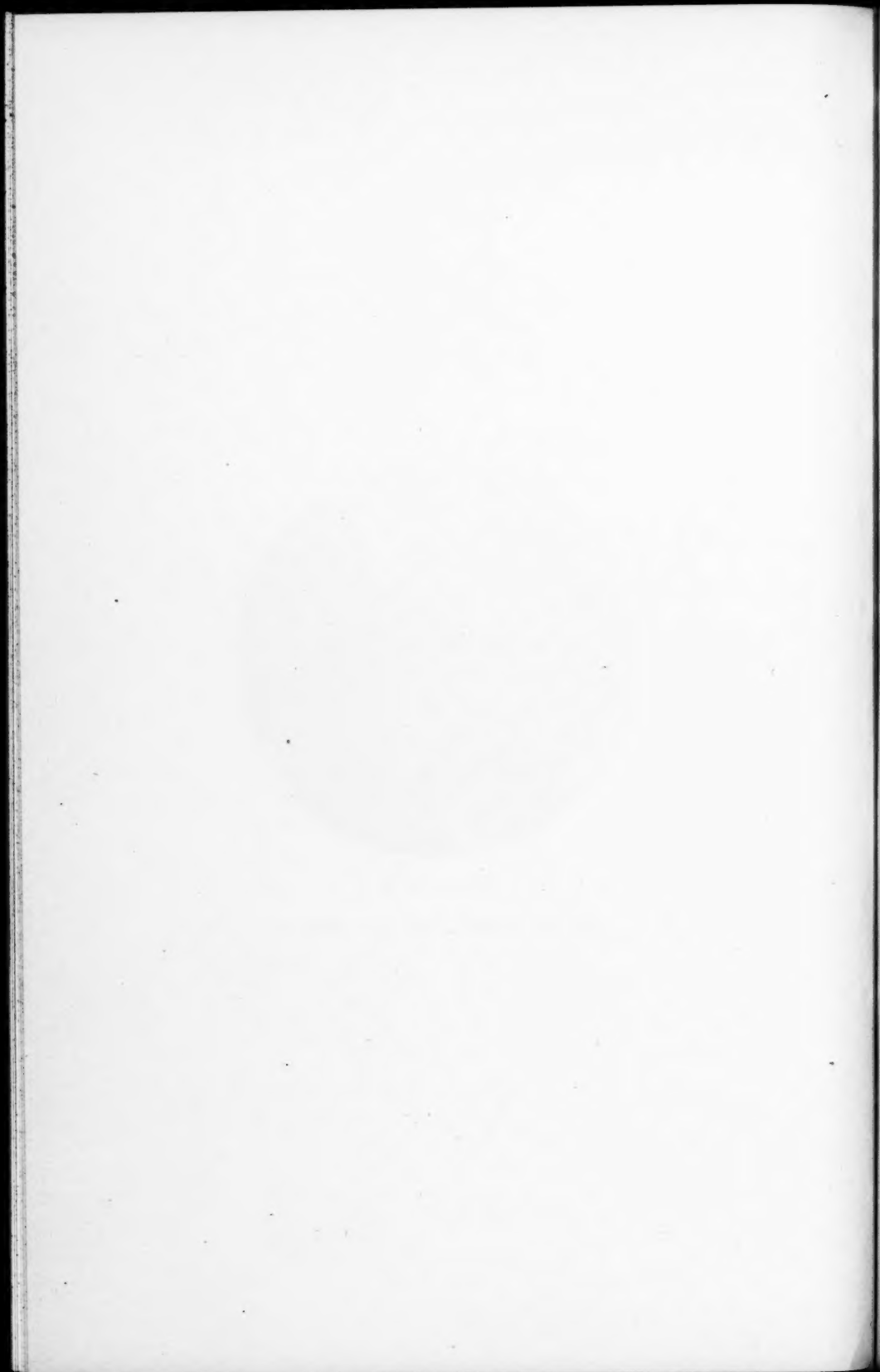
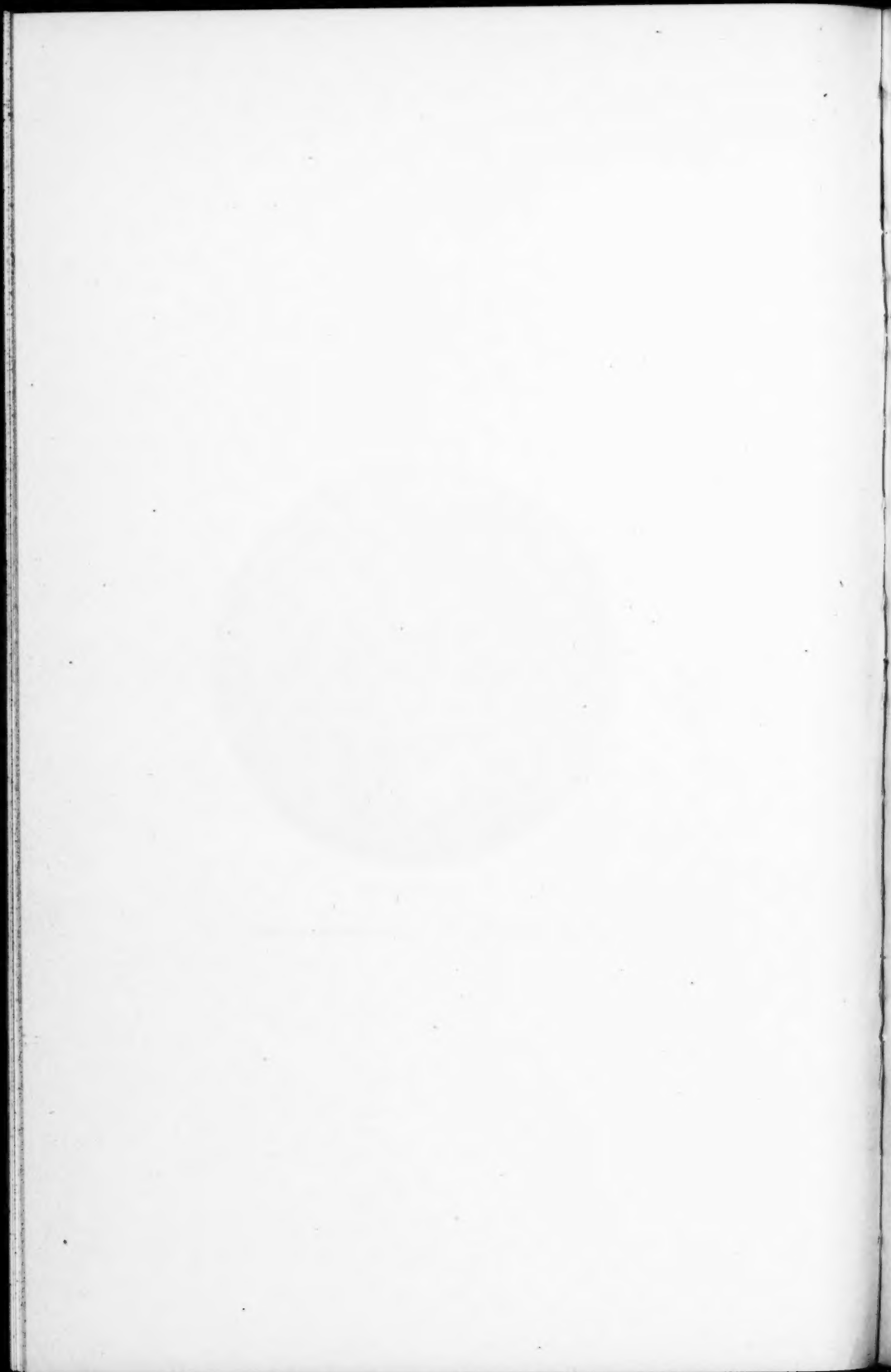




FIGURE XIX.

Showing relation of two tubercles to epithellum.



XVII.

SINISTER UNRECORDED ANOMALIES OF THE SPHENOID.*

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The two following specimens, in which diverticula of the mucous lining of the sphenoidal sinuses protruded directly into the subdural space, were evidently fraught with such serious possibilities to the lives of these individuals, that I felt justified in bringing them to the attention of specialists. Since both these individuals did not succumb to complications arising from these abnormalities, it would, however, be a mistake to attribute an altogether too sinister meaning to their presence. One of these individuals lived to the age of twenty-eight years, and the other to that of thirty years without complication from the unique defect. Nevertheless, it is evident that a sphenoidal sinusitis would probably have resulted in meningitis and death, and from the position of the defects and diverticula it is also clear that probing, irrigation or other operative procedures might have resulted disastrously through no lack of skill or foresight on the part of the operator.

It is to be hoped that such abnormalities are exceedingly rare, and the absence of references in the literature to similar defects would seem to indicate this. This seems particularly true since nothing wholly comparable to them was mentioned by Zuckerkandl, Killian, Onodi, Spee, Gibson, Schäffer or Loeb and others who have made a special study of the paranasal sinuses or of the sphenoid. Nor are such defects mentioned in the large handbooks on the nose and upper air passages, with the exception of the *Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege*, in which Onodi refers to Spee and Zuckerkandl.

*From the Division of Anatomy of the Stanford Medical School.

Perforate sphenoidal sinuses with subdural diverticula.

Specimen *a* is from the body of a white man, twenty-eight years of age, who died of tuberculosis. (See figure 1, *Spolia anatomica addenda*, Meyer, 1915.)

There are two sphenoidal sinuses, as usual, in this specimen, the right sinus extending several millimeters across the median line. The septum is located in almost a sagittal plane lying two to three millimeters to the left. The form of the right sinus is oval with its long axis, which is practically parallel to the upper surface of the basilar process of the occipital bone, making an angle of approximately forty-five degrees with the vertical. This diameter measures twenty-five and one-half millimeters, the vertical one fifteen millimeters, and the transverse (right to left) eighteen millimeters. The lining membrane of the sinuses are thin and not abnormally adherent anywhere. The roof of the right sinus is very thin, measuring only 0.35 millimeters (by micrometer caliper) directly beneath the sella and a little to the left of the median line. Its maximum thickness is only 0.5 millimeter.

Although the configuration of the left sinus is similar to that on the right, it is much smaller, measuring only eleven millimeters in a transverse (right to left) diameter and twenty-two millimeters in the longest oblique direction. It, too, is normal in appearance. The combined sinuses extend only about half way beneath the sella. The ostia are normal in size and position.

The nasal cavity is capacious but normal in appearance, and the conchæ, except the superior, are small. There are four conchæ on the left side, the posterior ethmoidal cell opening into the supreme meatus.

At about the midpoint of the ventral (anterior) portion of the lateral wall of the right sinus, immediately beneath its roof, there is an oval defect. The longest diameter of this oval defect measures seven millimeters and extends anterolaterally, lying almost in a horizontal plane and making forty-five degrees with a sagittal plane. The short diameter measures only 4.5 millimeters. Through this opening a diverticulum of the sinal lining, which is six millimeters long, protrudes into the subdural space. The wall of this diverticulum is very thin, nowhere adherent to the exceedingly thin (0.5 millimeter) but regular margin of the defect in the lateral wall, and can

be inverted with entire ease. It extends slightly forward and upward into a triangular space bounded by the optic nerve anteromedially, the carotid artery posteromedially, and the reflection of the dura laterally. The dura which surrounds the base of the diverticulum on all sides does not envelop the diverticulum or cover the defect in the lateral wall of the sphenoid, but merely comes into contact with the encephalic surface of the bony margins bounding the defect. Hence it is evident that the mucous diverticulum extends directly into the subdural space, and must have been in direct contact with the arachnoid.

There is no corresponding or other defect in the wall of the left sinus, but the corresponding region is marked by a depression which lies in the base of the posterior root of the lesser wing. This root is absent on the right side. The anterior clinoid process on the left side joins with the middle process, forming a complete foramen for the carotid artery. That on the right side had unfortunately been partly removed, but the condition of the middle clinoid process, which is wholly intact, shows clearly that it was not joined to the anterior clinoid process on this side. Furthermore, the lateral sinial wall with its dural reflection shows that the posterior root was absent on this side.

The anterior and middle ethmoidal cells and the frontal sinus are large, but the posterior ethmoid cells are extremely small, being three to four millimeters in size.

Specimen *b* is from the cadaver of a man, thirty years old, who died of tuberculosis. (See figure 1.)

The left sphenoidal sinus in this specimen is somewhat larger than the right, and extends completely beneath the hypophyseal fossa, being separated from the pons by an exceedingly thin bony wall only 0.27 millimeters thick. The hypophyseal fossa is large and long in a dorsoventral direction. The dorsum sellae is T-shaped in section, low (five millimeters high), and composed of a thin plate of bone which bears the large (by comparison) posterior clinoid processes. The floor of the sella forms the dorsal (posterior) half of the roof of the sinuses.

The ventral (anterior) half of the lateral wall of the left sinus contains a defect (see figure 1) similar in position and character to that in the preceding specimen. This defect is

oval also, but somewhat larger than the preceding, for it measures 6.5 x 4.5 millimeters. The bony margin bounding it protrudes slightly intracranially, forming a small cuff around the defect. This margin is only 0.25 millimeter thick.

The diverticulum of mucous membrane which protrudes through this defect extends fully 3.5 millimeters beyond the plane extending across the dural reflections over the optic and oculomotor nerves and the accompanying structures. (See figure 2.) It is slightly enlarged distally. Although the lining membrane of the sinus is somewhat thicker than that of the preceding case, it is nowhere adherent and could be removed very easily. The relations of the diverticulum to the surrounding structures are exactly the same as in the previous specimen. It has a total length of six millimeters with a width of seven millimeters in a line parallel to the optic nerve, and of 4.5 millimeters in a craniocaudal direction. Since a thin bony rim extends intracranially around the defect, for several millimeters, the optic nerve lies more above than medial to the defect. This bony rim also covers the ventral knee of the carotid artery.

The dorsoventral diameter of the left sinus is twenty-nine millimeters, and it extends several millimeters beyond the median line, which it intersects somewhat obliquely, the ventral half of the septum lying a trifle to the left of the median line. This sinus does not extend beneath the hypophyseal fossa on the right side except at its most caudal portion, although laterally to the right it extends beneath the middle cerebral fossa.

Just as on the left side in the previous case, there is a depression in the lateral wall of the right sinus in a position corresponding to the defect on the left side. As in the previous specimen, this depression was covered by the posterior root of the lesser wing, and in part also by a bony extension, not infrequently present, from the anterior to the middle and to the posterior clinoid process, which represents the ossified ligamenta interclinoidea. On the left side, on the contrary, there is no such bony union of the anterior and middle, but only of the middle and the posterior clinoid processes.

The finding of these defects in two specimens, neither of which was damaged by disease or injury, among only eight cadavers simultaneously under dissection, remotely suggests

John Burrows' dictum "That the number of birds one sees depends on the number one looks for." Not knowing of such possibilities, we had not looked for them. Nor can I believe that these instances are isolated cases, especially after having examined a small series of skulls with especial reference to the shape, form and position of the posterior root of the lesser wing and the varying osteology of the surrounding region. It is, for example, comparatively common to find a depression—i. e., an evagination of the sphenoidal sinuses—into the base of the posterior root; and in one cleaned remnant of a skull found in the laboratory, one of the sphenoidal sinuses communicated with the cranial cavity at exactly the same place as in the preceding specimens. Although the lesser wing of the sphenoid had been removed in this specimen, it was evident from the character of the margin of the defect in the lateral wall, and from the character of the wall itself, that it is not improbable that a defect was present in this specimen also before cleaning, for only a very slender posterior root could have been present. However, the mere presence of a very slender posterior root, or perhaps its absence, even, is not necessarily an indication that the sphenoidal sinus is not completely separated by bone from the cranial cavity. The absence of this root is probably only of significance if the sphenoidal sinus is as large or larger than normal—or, perhaps still better, only when there is a tendency for the sinus to extend laterally in the region of the base of the root. Just why absorption should be especially active here at the junction of the pre- and basisphenoids, I do not know, and it is possible, of course, that tension exerted through the root after fusion with the lateral wall may cause evagination of the sinus into the base of the root, or the root may be absorbed. This extension of the sphenoidal sinuses into the base of the posterior root of the anterior clinoid process is, of course, a wholly different thing than an extension of the sinuses into the lesser wings and anterior clinoid processes, said to occur in over eight per cent of skulls, according to Sieur and Jacob.

The absence of the posterior root in both the above specimens must leave a weak point in the wall at this region. Since the reflections of the dura over the carotid artery, and the second, third, fourth and sixth nerves lie at a higher intra-

cranial level, it is evident that the dura must at one time have been depressed over this region, in order to clothe the portion of the lateral wall later occupied by the defect. In pre- and early postnatal life there could, to be sure, have been no such dural depression, but with the change in contour and in the relation of the different portions of the sphenoid and the extension of the air sinuses with approaching maturity of the bone, such a condition was bound to appear. Since in these cases the lateral wall of the sphenoid was not reinforced by the posterior root, as is normally the case, it might be assumed that this region formed a point of least resistance to the developing and encroaching air sinus, were it not for the fact that reinforcements are absent on other portions of the sinal wall and yet no perforation or defects result. That the increased intrasphenoidal air pressure associated with such occasional phenomena as sneezing, coughing or forcible and obstructed expirations of any character, could be responsible for local atrophy of the bony wall and the overlying dura, seems decidedly unlikely, although the form of the diverticula and the bony margin of one of the defects seems to suggest that their form was modified by these things. But until we know more about the cause of the development of the air sinuses and the factors which control their extension in the different directions, it seems quite futile to speculate on the genesis of such defects.

It seemed highly probable to me at first sight that the dura must have covered these diverticula, but that it did not do so except at the very beginning of their extension through the bone is beyond question. Indeed, the perforation of the dura by the diverticula is the most interesting and unexpected thing. Moreover, it would have seemed likely that the cerebrospinal fluid and the other meninges and the brain substance might have forced these diverticula of mucous membrane back into the sinuses, or at least have prevented their protrusion through the dura into the subdural space. It would also seem as if one might have expected a diverticulum of the arachnoid, accompanied or unaccompanied—probably the former—by brain, to extend into the sinus as a result of the unopposed intracranial pressure. Moreover, since a defect was produced in the dura, it also seems as though the arachnoid should also have yielded to the same influences which caused perforation of the dura.

Unfortunately the brains had been removed from both these skulls, but the duræ were undisturbed in these regions. The presence of the protruding diverticula is conclusive evidence, however, of the fact that practically no pressure was exerted upon them, for their extremities were entirely unattached to the dura, and hence their inversion into the sinuses was prevented only by atmospheric pressure. Besides, had they been at all firmly attached to the arachnoid, it is more than likely that a tag of arachnoid would have remained attached to their distal extremities when the brains were removed. Hence it would seem that the arachnoid sealed the opening.

The only investigators who mention defects in the lateral wall of the sphenoid are Zuckerkandl, Spee and Onodi. Zuckerkandl (1893) spoke of having noticed physiologic dehiscences and small gaps in the lateral walls, and Spee speaks of small defects in the region of the sulci carotici in a juvenile skull. Since small defects in the lateral walls in the region of the carotid sulci—and in other places, for that matter—are not very infrequently seen in cleaned and dried skulls, it is evident that such specimens do not furnish proper evidence regarding the actual frequency of these abnormalities. If the walls are exceedingly thin, they are easily injured when the dura is stripped, and still more easily eroded in the cleaning. Hence, skulls in which the duræ have not been removed can alone be regarded as furnishing reliable evidence.

Onodi, who observed vascular sulci and foramina in the lateral wall, and also larger or elongated dehiscences in these vascular sulci, also emphasized the fact that besides being developmental anomalies, such dehiscences may be artifacts, or result from traumata or from senile atrophy or pathologic conditions. Onodi, who examined four thousand entire and several hundred cut skulls, reported no such defects as those found in the above cases. The same statement holds for Gibson (1908), who examined eighty-five specimens. And although Emerson (1908-1909) gave a "Report of a fatal operative case showing developmental absence of the outer sphenoidal wall, and in its place a large vein communicating directly with the cavernous sinus; autopsy," a review of the clinical history of this case clearly suggests that the long standing suppuration was very probably responsible for the absence of the lateral wall of the sphenoid.

According to the clinical history, a carious area was present in the sphenoid over three years before death, and a "carious area on the floor and inner wall were curetted" over two years before death. Furthermore, at the time of the fatal operation "a carious area was felt on the outer walls," which the autopsy revealed as having been developmentally absent! In view of these facts, and the further fact that Onodi and Zuckerkandl both emphasized the occurrence of physiologic dehiscences, the conclusion that there was a "developmental absence of the outer sphenoidal wall" in Emerson's case seems extremely unfounded.

Since the clinical significance of such defects in the lateral osseous walls, accompanied or unaccompanied by protrusions of the lining mucosa into the subdural space, must be evident to everyone, emphasis on this matter is unnecessary.

While examining a small series of skulls in regard to the position and size of the posterior root of the lesser wing of the sphenoid, in connection with the defects above reported, I was much impressed by the varying relations existing between the upper portion of the genu ventralis of the carotid artery and the optic nerve and the ophthalmic artery. Ordinarily, the dorsal or posterior margin of the posterior root of the lesser wing is located from two to four millimeters dorsal (posterior) to the similar margin of the broad anterior root which bounds the upper half of the optic foramen. But if the posterior root of the lesser wing is located far ventrally—i. e., anteriorly—it allows the genu ventralis of the carotid to lie far forward. In the specimen before me the posterior root lies four millimeters anterior (ventral) to the posterior or dorsal margin of the anterior root which forms the upper half of the optic foramen. Hence, it happens that the optic nerve and the ophthalmic artery come to lie directly upon the knee and a part of the upper turn of the carotid artery as the latter runs dorsally and upward to pass through the clinoidocarotid foramen.

Such a relationship between the carotid artery and the optic nerve and ophthalmic artery may be of far less consequence if the anterior root of the lesser wing does not extend dorsally almost as far as the base of the middle clinoid process, thus forming a roof over these structures. But when, as in the specimen under discussion, such an osseous roof is present, it

is evident that both the optic nerve and the ophthalmic artery are subjected to the full arterial pressure in the carotid, because the latter and the optic nerve and ophthalmic artery really lie directly inferiorly—i. e., caudal—to the broad anterior root.

In the ordinary or normal skull, both the optic nerve and ophthalmic artery lie upon the posterior root which is above the genu of the carotid artery. Hence, these structures are ordinarily protected from the direct effects of the arterial pressure in the carotid by the posterior root. Besides, since they are ordinarily not roofed over by an extension dorsally of the anterior root of the lesser wing, they cannot be forced against an unyielding structure.

In the specimen before me the foramen formed by the union of the anterior and middle clinoid processes, the clinoidocarotid foramen, is not bounded ventrally—that is, anteriorly—by the posterior root, as is usually the case, but by the margin of the broad anterior root. This ordinarily forms the upper anterior boundary of the optic foramen, and the posterior root forms the dorsal and inferior boundary. Consequently, in this specimen the optic nerve passed through two foramina—the optic and clinoidocarotid.

It is rather venturesome to draw inferences from the relationships here given, and I am reminded of the wisdom of the old saying: "Let not the cobbler go beyond his last." Yet one seems justified in asking whether the considerable pressures exerted in these cases upon the optic nerve by the carotid artery might not lead to optic atrophy as a result of direct pressure or of circulatory disturbances due to interference with the arterial current in the relatively small ophthalmic artery. Since the pressure in the carotid is exerted at right angles to the latter, such a result would seem to be inevitable unless the latter arises from the carotid unusually close to the optic foramen. The optic nerve could not escape this pressure, however; and since the pressure from arteriovenous aneurisms can cause interference with vision and optic atrophy in a comparatively short time, it is difficult to realize how the optic nerve could resist the considerable pressure in the carotid if exerted throughout the lifetime—or practically that—of an individual. It is also known that the comparatively temporary pressure exerted by the walls of the sphenoidal sinus, as a

result of distension by inflammatory processes, may cause optic atrophy. This fact, too, it seems to me, confirms the above supposition as to the possible effects of pressure from the carotid. My friend, Professor Rusk, also informs me that the pressure exerted on the base of the brain by extremely tortuous and sclerotic basilar arteries may cause symptoms of hemiplegia. Unfortunately, no operative procedures would be likely to reveal or could hope to relieve the conditions produced by these abnormalities.

The tuberculum sella (olivary eminence) is absent in this specimen, and the optic chiasmaticus very deep. The ventral or anterior wall of the hypophyseal fossa is practically vertical. The left sphenoidal sinus is spherical, and only about four millimeters in diameter. The right is oval, and measures about six millimeters in the longest axis. The ostia of these small sinuses were comparatively large, and opened almost directly downward. They were located in a plane directly ventral (anterior) to the posterior root of the lesser wing.

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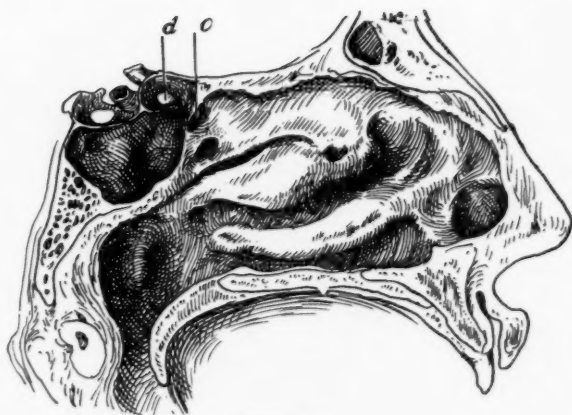


FIGURE I.

Lateral View.



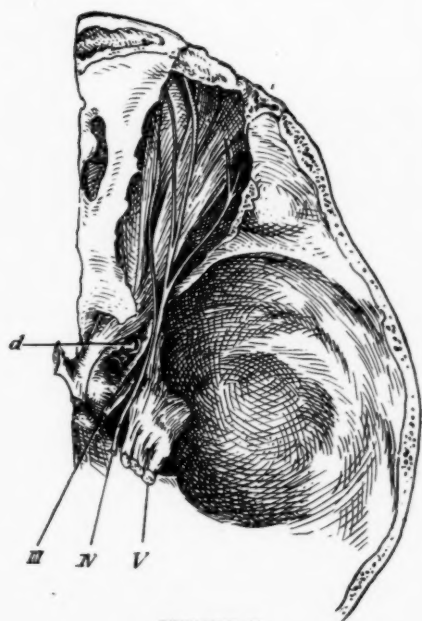


FIGURE II.

Superior View.

XVIII.

POLLEN THERAPY IN HAY FEVER.*

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BOSTON.

Within a few years numerous reports have appeared in regard to the treatment of hay fever patients by the subcutaneous injection of pollen extracts. The writers, among whom may be mentioned Freeman, Noon, Cook, Clowes, Koessler, Manning, Oppenheimer, and Gottlieb, have uniformly reported that a certain number of patients so treated have received more or less complete relief from the disease.

The object of the following paper is to report the results of observations based upon one hundred and twenty-two cases of hay fever examined during the past twelve months, with reference to determining, if possible, the value of this treatment, and also to ascertain what biologic relations, if any, exist between the pollen of different plants.

In the first place, a word should be said in regard to the method of obtaining and preserving the pollen extracts. For many plants, which furnish an abundance of easily detached pollen, it is sufficient to gather the partially opened flowers, bring them into a room without currents of air, and in the course of a few days the pollen may be shaken upon smooth paper. This applies particularly to those plants the cross fertilization of which is effected through the agency of air currents, such as many forest trees and grasses, and certain Compositæ, particularly ragweed. In the entomophylous plants, where the cross fertilization is largely effected by the agency of insects, the freshly opened anthers may be clipped and gathered. The pollen is then either placed in the solution for extraction, or it may be preserved dry for an indefinite period.

*Read before the American Laryngological Association, June 2, 1915, Niagara Falls, Canada.

I have taken pollen from specimens in my herbarium, gathered twenty-five to thirty years ago, of grasses and of ragweed, which on the addition of water excites as marked a skin reaction as would be the case with freshly prepared extract.

The extract is obtained from the pollen by soaking in water for a few hours. I have not found it necessary to subject the material to trituration, as advised by some writers; and this is also theoretically unnecessary, since the pollen grains in water promptly undergo a swelling, with solution of their albuminous contents. Several observers have complained of the difficulty of preserving the extract, and say that it is liable to deterioration on standing. After a number of tests, an alcohol dilution of thirteen to fifteen per cent seems to meet the requirements; and material prepared in this way a year ago has apparently lost little of its efficiency. It is interesting to observe, in this connection, that we have an example of a natural plant juice, namely, wine, in which preservation of its qualities is thus secured. The heavier natural wines contain a considerable amount of albuminous matter, together with approximately fourteen per cent alcohol. More than this percentage checks the further development of the yeast plant, and this amount, while preventing decomposition, does not seem to be sufficient to cause a precipitation of the proteids of the wine. It is desirable to keep the solution in amber bottles.

A word of caution should be said in regard to the gathering of flower heads for the preparation of pollen extract in the case of those plants which may contain a poisonous substance, as occurs in certain *Compositæ*, especially the wormwood group. I have observed a few cases where disturbing symptoms of nausea and malaise followed the injection of such material to a greater extent than would be accounted for by the actual amount of pollen present. In the case, however, of such plants as the *Rosacæ* and grasses, these precautions are unnecessary, and we may, in the case of the latter, find it more convenient to strip off the flower heads or anthers by hand.

When the pollen has been gathered and a suitable extract prepared, the latter constitutes then the stock solution from which varying dilutions are prepared. It is theoretically desirable to prepare a stock solution with a definite percent-

age of pollen extract; but practically this is unnecessary, since individuals differ very widely in their degrees of sensitization, and each case must be examined by different dilutions to determine the correct strength which it is safe to use for him.

As has been described by other writers, and by a previous publication of mine, the tests are made by making a series of superficial scratches on the skin of the arm and gently rubbing in a drop of the pollen extract to be tested into a scratch. After five to fifteen minutes the positive reactions are indicated by varying degrees of local disturbance. These disturbances of the skin may be ranged in order of intensity as follows: In some cases the first perceptible alteration consists in a sharply circumscribed white area, not elevated, bordering the scratch for a distance of one-sixteenth to one-eighth of an inch. We may find in other individuals the first manifestation to consist of a slightly reddened raised area. In more pronounced disturbances the area of swelling is more extensive and is more or less white in color, being surrounded by an area of reddening of varying size. When this degree of disturbance appears, it is usually accompanied by itching. It may in marked cases attain a considerable size, the edematous area reaching one inch or more in diameter, and surrounded by half an inch or more of hyperemia.

The intensity of the skin reaction does not always seem to be proportionate to the clinical symptoms of hay fever. I have seen numerous severe cases where the skin reaction was much less than in other individuals, who apparently suffered from a milder form of the disease. In the case of children, the skin disturbances are relatively less pronounced than in the case of adults, and I have observed several under ten years of age, with apparently well defined hay fever, who showed no reaction to the prevailing pollens borne in the air at the special season.

Examination of my cases by this method shows that the hay fever season in eastern North America may be divided into four periods, as follows: The first period coincides with the flowering of the earliest blooming plants, the second with the flowering of the grasses, the third with the midseason flowers of July, and the fourth with the opening of the autumn blooming Compositæ.

Of the first period, thirteen patients were observed who gave positive reactions to one or more of the following plants:

Coltsfoot (*Tussilago*).
Dandelion (*Taraxacum*).
White maple (*Acer dasycarpum*).
Willow (*Salix nigra*).
Alder (*Alnus incana*).
Birch, white, yellow and black (*Betula papyrifera lutea*
and *lenta*).
Hawthorne (*Crataegus* sp.).
Apple (*Pyrus malus* and *floribunda*).
Lilac (*Syringa* var.).
Oak (*Quercus rubra*).
Tulip.
Lily of the valley.

The symptoms which these early bloomers excite are relatively mild, and depend chiefly upon the abundance of the plants in the vicinity of the patients. For many individuals the annoyance is so slight as to be perhaps not worth treating. In others, a residence surrounded by maples or oaks, or with an orchard close at hand, may cause considerable discomfort. The most severe cases of this season which I have seen were from maple and oak. If there is only one sensitization, the period of disturbance is brief and disappears with the drying or shedding of the flowers in question.

The second period, represented by thirty-six cases, is ushered in with the flowering of the grasses. Owing to their abundance and the range of the blooming period of the different species, it is relatively severe and prolonged. Coincident with their flowering we may find an associated disturbance from many garden flowers, but for the majority of individuals, these latter are of minor importance. It is probable that if these June cases can be rendered immune to grasses, the other causes will be borne without much trouble.

The third period, represented by ten cases, has been shown by my observations to be due in great measure to the mid-season Compositæ, such as field daisy, hawkweed, yarrow, etc. With these are many species reported by patients which are still to be studied, such as chestnut, phlox, mountain laurel, and numerous other plants of restricted distribution.

The fourth period, with seventy-seven cases, begins with the general flowering of the ragweed, goldenrod, asters and the late Compositæ, and lasts until frost. My examination of the fields and roadsides at this season shows that the common plants other than the Compositæ are relatively innocuous and may be disregarded. Such are, in the vicinity of Boston, the wild carrot (*Daucus carota*), pigweed (*Chenopodium*), hardhack (*Spiraea tomentosa*), and other representatives of these families.

From the foregoing observations it is evident that in this vicinity the chief causes of hay fever are the grasses and the late Compositæ. The early and midseason forms are briefer and less severe.

The possibility has been suggested that sensitization to proteids may arise through some parenteral entrance of the albumen in question into the body, and that hay fever, for instance, may originate from the contact of a given pollen with a scratch or abrasion of the skin. If this should be the case, it would certainly lay the method of testing by skin reaction open to serious objection, as we should thereby run the risk of exposing the individual to numerous sensitizations. I have gone over this point carefully with many patients, testing them repeatedly with pollens to which they were originally negative, and have not, in any case, discovered the subsequent development of a sensitization.

METHOD OF TREATMENT.

The dosage is determined in the following way: After the special exciting pollen has been ascertained by the skin test, a second series of scratches is made at a distance from the first, and different dilutions of the pollen extract in question are applied. It is important not to have this test applied in the vicinity of the skin which has been reddened by the first tests, as an increased excitability of this region is present, and even a simple scratch will cause localized swelling. The dilutions may be most conveniently made by adding a certain amount of the stock solution to alcohol of the same strength, and a twenty-five per cent, ten per cent, one per cent, and even weaker, dilution of the original extract are applied to the second series of scratches. The initial dose is determined by the dilution which fails to excite a definite skin reaction,

and for the sake of causing as little smarting from the alcohol as possible, the quantity of material injected should not exceed five or ten drops. I should also recommend postponing the injection of even a small amount until after the reaction from the first skin tests have subsided, since a considerable amount of absorption from these probably takes place, and the introduction of an additional amount has seemed to me in a few cases to cause systemic disturbance. The injections may be made at intervals of two days to a week, increasing by a few drops at first, and later by the absorption of stronger percentages of the stock solution. To avoid the risk of anaphylactic disturbance, I have advanced the strength at first slowly. After five or more injections have been given, the strength may be increased with greater rapidity. The disturbances occasioned by the injection of the pollen extract below the skin (not intramuscular) consist, when a sufficient strength has been attained, of a more or less well defined lump, varying from the size of a bean to that of a pigeon's egg, accompanied by a sensation of moderate heat and itching. It is interesting to note that none of the patients have complained of these manifestations as representing more than a trivial degree of discomfort, and in no case has the disturbance been sufficient to cause the patient to interrupt treatment.

Two instances of distinct but harmless anaphylactic shock were observed, one occurring in August in a man who came in somewhat exhausted from the heat, who showed extremely marked skin reactions, and who received on the same occasion a small dose of ragweed pollen extract. About half an hour after the administration of the pollen extract, he was taken with faintness, nausea, and vomiting, and had to be assisted home. The other case was that of a boy who received a dose of grass and dandelion pollen insufficient to cause local skin reaction, but who a few hours later was taken with moderate prostration, nausea, and vomiting. It has seemed to me consequently a wise precaution to make the initial dose approximately one-tenth of that which the patient can theoretically receive with safety. After a number of injections have been given, it is practicable and safe to inject a strength which may cause distinct skin reaction, without exciting general disturbance.

Beyond the immediate anaphylactic shocks above noted, I

have seen in a few cases where ragweed pollen has been injected occasionally a more or less marked persistent general depression. Since using the pure pollen extract, such a condition has not occurred in my own practice, but it has been reported to me by patients treated by other physicians. It seems to me here to be a question whether there has been an injection of toxic material other than the pollen, or whether the dosage has been excessive.

RESULTS OF TREATMENT.

We have two methods of determining the effects that have been accomplished by the injection of pollen extracts: First, the behavior of the skin reactions; second, the patient's observations regarding his condition. The first phenomena, being objective in character, should afford a more reliable guide as to what has been accomplished.

INFLUENCE OF TREATMENT UPON THE SKIN REACTIONS.

In those cases which it was possible to observe for a period of two weeks to several months, the skin reactions were carefully noted at regular intervals. Of these, sixty-two showed a distinct diminution in the size and intensity of the disturbances in the skin, two showed no appreciable change.

The time required for the diminution of the skin reaction varies within wide limits, dependent both upon the plant tested and upon the individual himself. In the case of alder, willow and apple, I have sometimes obtained a marked reaction from the first application, but on repeating the application two days later, a diminution or total disappearance was noted. This sudden disappearance following the first scratch was noted also in a case of horse fever, which showed on one afternoon a very extensive swelling from the application of antitoxin to a scratch, but who on the following morning, tested with the same antitoxin, showed no reaction, and has shown none since. In the case of grasses, and even more in the case of ragweed, a slower disappearance of the reaction was noted. With ragweed and cosmos particularly I have observed in some cases a persistence of the skin reaction, even after weekly injections carried on for a period of four to six months. In these more obstinate cases an increased tolerance, however, was noted in spite of the persistent swelling. This tolerance was shown by the disappearance of itching which

the first injections had caused, and by the smaller amount of hyperemia surrounding the edema.

Twenty-one cases of those whose reactions had diminished in the course of treatment were examined again after the lapse of two to six months. Of these, nineteen showed no return of the skin sensitiveness, and some of them showed a still further diminution. Three cases after the lapse of three months showed a distinct return toward their original degree of sensitization.

INFLUENCE OF TREATMENT UPON THE HAY FEVER SYMPTOMS.

In describing the results which have followed the injection of pollen extracts, it has seemed to me desirable to separate those cases which have received treatment during the hay fever season from those which have been treated during the winter or out of season. I have done this for the reason that it is difficult during the course of an attack to draw accurate deductions from the statements of the patients themselves. We have, in the first place, to remember that seasons vary in the severity of hay fever symptoms, dependent upon the amount of rain, heat, and cold. Furthermore, the individual's predisposition seems to vary, perhaps as the result of his physical state and habits at the season. Finally, the element of suggestion may conceivably play a part. While I shall, therefore, report the summer cases with reference to the degree of relief obtained, I do not regard these figures as at all conclusive. On the other hand, observations carried out during the winter with reference to changes in the intensity of the skin reaction, may be considered a fairly reliable guide, if it be admitted that the strength of the solutions themselves has not undergone deterioration. This latter point is difficult to determine with absolute certainty. Nevertheless, my alcoholic solutions seem now, after the lapse of months, to effect in new cases nearly, if not quite, the same degree of skin reaction which they occasioned when in a fresh state.

At the date of writing, one hundred and twenty-two cases have been observed, of which seventy-four have had more or less treatment, and forty-eight have been seen but once, or are now beginning treatment. Of the cases which may have been considered to have had a sufficient amount of treatment to enable us to draw more or less definite conclusions, thirty-two were treated after the onset of the hay fever symptoms,

and forty-seven were treated during the winter or early spring. Of those cases which were treated either at the beginning or immediately before the hay fever season, twenty-six expressed themselves as having been more or less relieved, eight could not see material improvement. In estimating the results actually achieved by treatment during the season, I believe that an accurate judgment would place the extent of the relief in a number of instances distinctly below that which the patients expressed. In other words, I believe the element of suggestion plays here a considerable part. Furthermore, a certain number of these who believed themselves improved showed but slight diminution in the extent of the skin reaction. It has seemed to me that such cases probably represent too high a degree of sensitization to obtain material relief during the hay fever season, and that a longer period of treatment is required. On the other hand, several of these individuals who reported some months later, showed a marked diminution in their skin reaction, and it was possible then to undertake their treatment with the result of bringing about still further a diminution in the intensity of the skin disturbances. About one-fourth, however, of those treated during the season experienced after a certain number of injections, ranging from four to twelve in number, such striking diminution in their subjective sensations and in the skin reactions, that it seemed difficult to ascribe the gain to anything else than the treatment, the improvement noted having occurred from one to three weeks before the disappearance of their type of hay fever in this vicinity.

THE BIOLOGIC RELATION BETWEEN DIFFERENT PLANT PROTEIDS
OR POLLENS.

It is evident that in this work a knowledge of the biologic relationships of the exciting plants would be of the greatest aid. If, for instance, we can say that the protein of two exciting plants is identical, we shall need to inject the pollen of only one.

While we do not know clinically the relation of the different plant proteins, yet from a botanic standpoint they have been studied by serobiologic methods by numerous observers.*

**Physiologische-systematische Untersuchungen ueber die Verwandtschaften der Angiospermen.* Mez und Gohlke, Cohn's Beitrage z. Biologie der Pflanzen, 1913.

The methods employed were the precipitation reaction and the agglutination method. Extracts of the plant albumen were made, removing first, where present, fats, oils, acids, alkaloids, starches, glycogens, and sugar. These extracts were then injected into rabbits, preferably into the abdominal skin. As a rule, the interval between injections was from three to four days.

With reference to the time required for immunization of the rabbits, no exact figures can be given. Sometimes a potent immune serum can be produced after three to four injections, but cases also occur where, even after ten injections, very little immunity had appeared, and at times entirely failed to occur. Apparently the individuality of the animal is a factor. Uhlenhuth mentions that of ten rabbits injected with the same albumen, only one showed a potent immune serum. The test is done by removing a small amount of blood from the marginal vein of the rabbit's ear, adding a portion of the extract which was used for injection, and centrifuging. If the serum is of high potency a precipitate is shown. The animal is then separated from the others, not fed for twenty-four hours, and then killed. The blood is received in sterile glasses from a carotid incision. The serum must be perfectly clear and show no opalescence. Finally, the serum must have no free antigen—that is, the animal must not be killed too early after the last injection. The serum, if absolutely sterile, is easily preserved in dark glasses and closed with sterile cotton.

Precipitation Method.—Dilutions of the plant extract are made, beginning with 1 to 200 and ending 1 to 50,000. To each of these dilutions a cubic centimeter of serum is given. After keeping in the thermostat for an hour at 37° centigrade in the more dilute preparations, there is a precipitate which demonstrates the value of the serum. The procedure in the case of material related to the original one is analogous. A precipitate shows a relationship, the absence of precipitate indicates that relations of identity with the original albumin are not present.

Agglutination Method.—Here one places in different glasses the same dilution of the extract, according to the content of albumen, for instance, 1 to 200, and to these glasses different degrees of immune serum are added, 8/100 of a centimeter,

2/100 of a centimeter, 1/100 of a centimeter and 5/1000. These mixtures are then sensitized for two hours in the thermostat, and after the lapse of this time, $\frac{1}{10}$ of a cubic centimeter of fresh, active ox serum is added, whereupon, in the case of related albumen, coagulation occurs. This method has shown itself particularly adapted, provided we have a specially potent immune serum. In any serum the two methods should be employed before important results are accepted.

It is desirable in these tests to bring the solution to the same content of albumen.

A large number of families have been already studied with regard to their position in the system. It has been shown that the development of the angiosperms extends from the Selaginellaceæ over the pines towards the magnolias, that the yews divide laterally from the pines, while the Gnetaceæ form another lateral branch of the conifers, and then the trunk runs from the barberries towards the roses and ends with the myrtles. The magnolias and barberries belong to the common stem of the roses and crucifers. The trunk must take a branch below the Ranunculaceæ, of which the ends are the crucifers and the roses. In phylogenetic respect the barberries are older than the buttercups. Likewise the pea family are closely related to the roses. The trunk then proceeds from the roses to the Crassulaceæ and saxifrage family, and ends with the evening primrose and myrtles. The mignonettes and Capparadaceæ lie between the barberries and mustard family. Since the albumen of the mignonette and Capparadaceæ reacts like the albumen of the poppies, it is possible that the branch of the stem between the magnolias and Capparadaceæ takes place above the mignonettes. Poppies do not act reciprocally with the mustard. The mignonettes, however, stand close to the violets. The teagle family, Dipsacaceæ, which apparently resembles the Compositæ, does not react with it, and we have here to do with an interesting case of convergence.

The accompanying diagram shows the mutual relationships of the families which have been studied by their serum reactions. Unfortunately, the position of the grasses is not given in this diagram, as they belong to a separate trunk, namely, the Monocotyledons, but this fact renders it sufficiently evident that their albumen is wholly distinct from that of

the dicotyledous. It has, however, been established that they are closely related in their reactions to the Liliaceæ.

From the foregoing considerations it is evident that the families which chiefly concern us in this present connection, namely, the Gramineæ, the Compositæ, the Rosaceæ, and certain families of trees, have no serobiologic affinity with each other. Consequently, an individual sensitized to one family alone would not react to the pollen of a distant group. Where two families are closely related, as in the Rosaceæ and Leguminosæ, a positive reaction might be expected to both. The clinical evidence in this regard extends as yet only to the Gramineæ and the Compositæ. Clowes showed in 1913 that in individuals sensitized to both grasses and ragweed, immunization against the former conferred no immunity against the latter. Much consequently remains to be studied in this respect.

In the case of genera within a given family, a relationship of proteids has been shown to exist, as already stated. We should, therefore, expect an individual suffering from ragweed sensitization to react positively also to goldenrod and to the other members of the Compositæ. A case which reacts positively to beach grass would also react to June grass, red top, etc. A case reacting to roses would react to apples, cherries, spirea, etc. We have, up to the present time, clinical confirmation of this view only in the case of grasses. Noon and Freeman showed that injection with one species of grass would also protect against other grasses studied. While they found the pollens identical, yet some grasses seemed to yield a more active extract than others, and in their later work used timothy grass by preference. My own study of the skin reactions in the case of grasses confirms this view, and, as I have previously reported, patients sensitized to one species react positively to others, although one may bloom in June and another—e. g., beach grass—in August.

Sensitization to the Compositæ represents for us in America the most important forms of hay fever. This is sufficiently shown by the generally accepted opinion of the majority of sufferers, and is confirmed by my own statistics. The question, however, has remained as yet unanswered, whether among the compositæ themselves subdivisions exist of a serobiologic nature. While we may admit a sufficiently definite

affinity to cause positive precipitation or agglutination reactions among all its members, yet it is conceivable that the proteid of the *Compositæ* may be still further differentiated in a manner corresponding to the different genera. Following the same hypothesis, we may imagine an individual sensitized primarily to the goldenrod proteid, who may show only in a relatively minor fashion sensitization to ragweed, with which he has perhaps never previously come in contact. He may consequently show marked reaction to the former and but slight reaction to the latter, yet in virtue of the underlying biologic affinity of the two proteids, immunization to the one may confer resistance also to the other.

During the past year I have studied ten species of the *Compositæ*, representing as many genera, in an effort to obtain light upon this subject. Two methods have been used: first, the classification of patients with reference to their reactions or preponderance of intensity of their reactions to the pollen of these *Compositæ*; second, observation whether injection of one species of pollen would influence the skin reactions to other pollens.

I.—CLASSIFICATION OF REACTIONS.

A summary of the relative frequency of the various sensitizations is as follows:

Ragweed	82 cases positive, 24 negative
Goldenrod	48 cases positive, 50 negative
Cosmos	28 cases positive, 25 negative
Field daisy	28 cases positive, 37 negative
Hawkweed	20 cases positive, 39 negative
Yarrow	21 cases positive, 28 negative
English daisy	16 cases positive, 19 negative
Tansy	11 cases positive, 20 negative
Coltsfoot	5 cases positive, 2 negative
Dandelion	6 cases positive, 1 negative

The disparity between these figures immediately suggests the question whether the different test solutions contained proteids of different strength, so that individuals with but slight sensitization did not react to certain weaker solutions, when they might have reacted to stronger ones. That this is not the case seems to be shown by the following table of twenty illustrative cases:

TABLE I.

Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Ragweed	4	3	3	4	4	0	3	3	3	3	4	4	4	..	1	3	1	2	3	4
Goldenrod	1	2	3	0	0	3	0	2	3	4	2	1	3	0	1	1	0	0	4	1
Cosmos	0	4	0	..	4	0	4	4	4	4	1	0	4	..	1	3	3	0	0	1
Field Daisy	0	3	0	0	0	0	0	2	0	0	0	2	2	0	1	0	0	3	..	1
Hawkweed	0	3	0	0	0	0	0	2	1	3	1	..	3	0	1	0	0	1	0	..
Yarrow	0	..	0	0	0	2	0	1	1	2	2	0	1	0	0	3	0	0
English Daisy	2	..	3	2	1	..	1	1	..	3
Tansy	0	0	0	2	1	..	2	..	4	..	0	..	0	..	0	0
Coltsfoot	4	4	4
Dandelion	4	4	3	..	1

The intensity of the skin reactions is shown by the numerals: 0—Negative, 1—Slight, 2—Moderate, 3—Considerable, 4—Marked.

From the table it is evident that all manner of variations exist in the primary or preponderating sensitizations of individuals. I have been particularly struck by the difference in the grouping of reactions exhibited by patients coming to me from different localities. As yet sufficient data are not at hand to enable me to generalize in this respect, but experience so far has led me to expect a different display of reactions in a patient of my vicinity from that shown in a patient from western New York or the middle West.

II.—INFLUENCE OF TREATMENT UPON REACTIONS OF RELATED POLLENS.

The second method of determining the relations existing between the proteids in the related genera of the Compositae, has been by the following method: A patient which shows sensitization to, for instance, ragweed and goldenrod, is injected in the usual manner with ragweed pollen and observations are made at intervals with reference to the goldenrod reaction. In some of these cases so studied it has been evident that with the diminution in the intensity of the ragweed reaction there is a diminution likewise in the goldenrod reaction, which, however, follows at some distance behind it, until the ragweed reaction has nearly or quite disappeared, when there is a sudden drop in the goldenrod reaction. My observations in regard to this point are as yet incomplete and demand further study before they can be regarded as conclusive. There seems, however, here a promising field for study, and the method is suggested for the consideration of investigators who are entering upon this work.

If these observations are confirmed, they will serve to indicate that there are slight differences between related genera and yet a fundamental relationship.

From the theoretic point of view, it would follow that while we may be able to accomplish immunization to all members of a given family by the injection of pollen from one, yet more rapid results may possibly be attained by employing a mixture of these pollens to which the individual shows preeminent sensitization.

The question may possibly be raised whether the parental administration of plant proteids may cause harmful alterations in the organs of the body. Longcope has shown

that nephritis may be set up in animals by the subcutaneous injection of egg white. No examinations have been undertaken, so far as I know, on hay fever patients, as to whether the injection of pollen extract has been followed by the occurrence of albumin in the urine. I have made no examination of my hay fever patients in this regard. A case of horse asthma, which I have reported elsewhere, received for a period of several months weekly subcutaneous injections of horse serum, in doses which finally reached the amount of five drops at each injection. The urine in this case was examined before and after treatment with negative results.

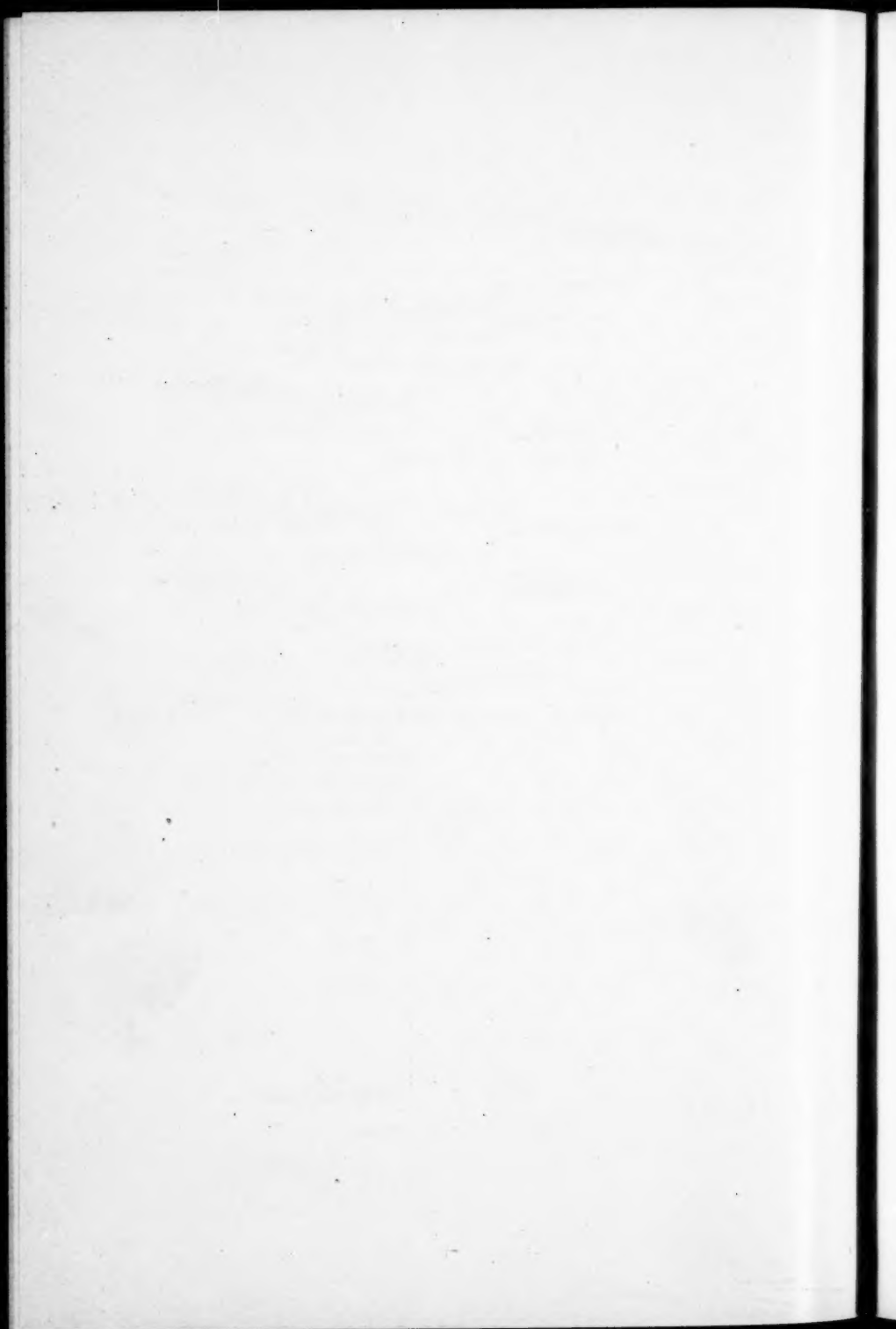
CONCLUSIONS.

Serobiologic methods have shown the phylogenetic relationship of the different plant orders and families. The application of these discoveries to the treatment of hay fever by injection of plant proteids promises to assist in the selection of the specific material required for a given case.

Definite reactions are elicited in hay fever by the pollen of the exciting plants when brought into contact with an abrasion of the skin. The intensity of these skin manifestations may be sensibly diminished by the repeated parenteral administration of the proteids in question. Coincident with the diminution in the skin reactions there seems to occur an increased tolerance of the exposed mucous membranes to the pollens of the plants employed. Pollen therapy in hay fever may be regarded at the present time as a promising method of treatment, but its value and the permanence of its results remain still to be definitely established.



After Mez and Gohlke.



XIX.

EXPERIMENTS WITH AUTOSERUM IN THE TREATMENT OF HAY FEVER. THE RELATION OF ANAPHYLAXIS AND EOSINOPHILIA TO HAY FEVER. A GENERAL SURVEY OF NEW METHODS OF TREATMENT.*

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Within the past year a new advance has apparently been made in the treatment of various persistent and chronic dermatoses by the use of autoserum. Among those who have been doing a large amount of work in this direction are Dr. Wm. S. Gottheil and his associates. Gottheil¹ in April of this year published his first report, which was followed later² by a more lengthy survey of the subject.

In this report the writer speaks enthusiastically about the beneficial changes that take place, particularly in the cases of psoriasis. All of the cases treated were of long standing, with extensive lesions, in which the usual treatments were of no avail. Gottheil summarizes his experiences with autoserum by stating "that it has a peculiar influence for good which enables us to clear off the skin in one-tenth the usual time, with very weak and innocuous local medication, and without any internal treatment at all." His results have been corroborated by Dr. John A. Fordyce and Dr. Howard Fox.

As a result of the encouraging reports in the treatment of the dermatoses by autoserum, it occurred to the writer that it would be worth while trying this same form of treatment in cases of hay fever. He arrived at this conclusion after noting the similarity of hay fever to various skin conditions. His premises may be summarized as follows:

1. Hay fever is a local manifestation of some internal derangement of the system.

*Candidate's thesis, accepted by the American Laryngological, Rhinological and Otological Society, December, 1914.

2. Hay fever has associated with it a nervous element which results in a local paresis of the blood vessels of the nose.

3. Hay fever occurs at well regulated intervals, and mainly affects definite parts, namely, the mucous membrane of the nose, eyes, throat, and lungs.

a. Psoriasis (with some others of the dermatoses) is a local manifestation of an internal derangement of the system.

b. Psoriasis has associated with it a certain nervous element.

c. Psoriasis occurs in definite localities on the skin (mainly extensor surfaces).

This line of reasoning was further substantiated by reasoning along biologic grounds, for (1) there are certain anaphylactic reactions present in psoriasis and other dermatoses which are at the same time present in hay fever and asthma. (2) A marked eosinophilia is present in the dermatoses and is also present in hay fever and asthma.

It was not, therefore, inconsistent or unreasonable to suppose that if a definite improvement or even cure could be obtained from the use of autoserum in the dermatoses, it could be obtained in hay fever and asthma.

The writer, therefore, began a series of experiments in August of this year, waiting until the patient was in the prime of his hay fever attack before beginning the injections.

Autoserum, as the name indicates, is a serum obtained from the patient into whom afterwards it is to be injected. The method of obtaining this serum is very simple, and is as follows: A constricting bandage, preferably of rubber, is wound around the patient's arm above the elbow until it is tight enough to almost obliterate the pulse. In a few moments the arm becomes a dull purple from the venous engorgement, and a vein from which to draw the blood is readily selected. Very often the very superficial veins are too small, and a deeper one must be palpated for. The desired amount of blood may be withdrawn in a number of ways. At first the writer used the MacRae needles, as suggested by Dr. Howard Fox. These needles are very small and are fitted into a rubber cork which can readily be inserted into a fifty cubic centimeter bottle. On one side is a thin narrow canula, to which a piece of rubber tubing may be attached for suction. It is claimed that after the blood once starts to flow, a continuous stream may

be kept up by proper suction through the rubber tube; but it was the writer's experience that on account of the small caliber of the needle it became clogged with coagulated blood very readily, so that it was difficult to withdraw all the blood that one desired. He therefore had recourse to the simpler (and what seemed to the patient more barbarous) procedure of using a twenty-five cubic centimeter all glass syringe to which could be attached needles of suitable length and caliber. He was able in this latter way to withdraw as much as twenty-five to thirty cubic centimeters of blood.

The blood was immediately projected into a sterilized fifty cubic centimeter bottle, made of a suitable size so that it could be used in an electric centrifuge.

This blood was then centrifuged at great speed for from twenty to twenty-five minutes, at the end of which time it was seen that the serum had been nicely separated from the red blood cells. It should be straw colored in appearance, but very often a small amount of the coloring matter of the blood tinges it, which apparently does no harm.

This serum was then drawn up into a sterilized all glass syringe and reinjected into the patient, either subcutaneously or intravenously. The writer found the former method satisfactory, and in no instance was there any untoward results. A large swelling often appeared at the time of the injection, but this disappeared within a few hours.

In a few instances when the centrifuge was out of order, it was necessary to allow the blood to stand over night in the ice box. The separation under these circumstances was just as good, but whether any chemical change took place, it is impossible to say.

In this series of experiments twelve cases were treated. The treatments were given in the writer's office, under the best conditions possible. Each patient was intelligent enough to let him know in a satisfactory way just what results there were from the injections. At first there seemed to be a period of vast improvement, and he was in hopes that we were at last on the road to a discovery of some specific cure. However, the improvement lasted but a very short time, and then the patients were just as bad as ever. It is, therefore, necessary to report these experiments from a negative point of

view, in the hope that it will lead others to do further research work along the lines of experimental biology.

It will be noted in the cases which are reviewed that the amount of serum injected was very much smaller than that used by the skin specialists. Gottheil in his later paper speaks of one case where he withdrew as much as one hundred and fifty cubic centimeters of blood, from which he obtained seventy cubic centimeters of serum. In these cases the largest amount of serum injected was fifteen cubic centimeters, but it seemed that it should have been possible to obtain a reaction of some sort from this small amount, if the treatment was going to do any good at all. Again, it is possible that in the treatment of the dermatoses the injection of such large amounts of serum was not necessary. As the susceptible hay fever patient is very sensitive to an infinitesimal amount of pollen, it is not unreasonable to suppose that moderate amounts of serum ought to show some reaction, if the treatment were to be worth anything at all.

It is worthy of note in the series of cases under observation that most of the patients had tried many of the newer treatments for hay fever, and in no instance was even a semblance of a cure effected.

A few of the cases are detailed below:

Case 1.—Mrs. K. First attack of hay fever nine years ago, returning each year and lasting from about the 15th of August to the end of September. She tried no cures except "cold" remedies until this year, when she was given hypodermic injections of some serum two or three times a week during the hay fever period, but with no benefit. After the season was over she was in a general run-down condition.

On August 17th this patient had ten cubic centimeters of blood withdrawn; on August 19th, nineteen cubic centimeters; August 21st, twenty-five cubic centimeters, and August 25th, thirty cubic centimeters. At the first sitting five cubic centimeters were injected subcutaneously in the arm; on the 19th, twelve cubic centimeters; on the 21st, ten cubic centimeters were injected into the buttocks, and on the 25th, eight cubic centimeters were again injected into the arm, after the blood had stood in the ice box for twenty-four hours. During the first week the patient seemed considerably improved, but after that time there was a retrogression, and the patient was as

badly off as ever. This patient has a general hypertrophy of all the mucous membrane of the nose.

Case 2.—Major D., whose hay fever dates from 1889. He found that when he was away from the United States he had no hay fever, but if he returned to this country in the summer it would be just as bad as ever. He had been stationed in Washington, West Virginia, Portland, Oregon, and other places, and in each place had hay fever. He had tried atropin, adrenalin chlorid and sprays of various kinds, with no relief.

Twenty cubic centimeters of blood were withdrawn on August 17th, half the amount in serum being reinjected, and the procedure repeated on August 19th. The patient then left for Porto Rico. His statement of his symptoms was as follows: August 18th he was worse; there was some discomfort most of the day. After the second treatment, until night time he seemed considerably better, but he woke up with a sensation of swelling in the right nostril. On the 20th he had very little discomfort all day. On the 21st he had an attack lasting for two hours. On the 22d he sailed for Porto Rico, arriving there on the 27th of August with apparently no symptoms.

In the conclusion of the report the patient says: "Comparing this attack with my best recollections of previous attacks, I should say that the attack I had on the night of August 19th was similar to a previous one six years before. To the best of my judgment the attack this year progressed at its usual rate, and was in no manner retarded by the two treatments administered."

Case 3.—Mrs. F. had her first attack of hay fever in 1908. Adrenalin was prescribed with no result. A new attack came on while the patient was in England the following year. Walking in the open air seemed to help. "This year I have worked in my garden and played tennis, always either to ward off an attack or more often to cure one."

The patient received two injections of ten cubic centimeters, from twenty-two cubic centimeters of blood withdrawn. The injections had apparently no beneficial effects.

Case 4.—G. L. B., adult. Has had attacks of hay fever for seventeen years, from the middle of August to the latter part of September. This had been associated with some

asthma. To quote the patient: "I have used at different times douches of Dobell's solution, peroxid, alkalin washes and adrenalin, coryza snuff, medicated smoke inhalation and camphorated snuff, all of which aided in clearing out the nose, insuring free breathing, and enabling me to get some sleep. This year I am using with good results camphorated snuff and a liquid to rub on the chest and veins leading to the head. Before retiring I place a small piece of gum camphor in my mouth which enables me to fall asleep."

This patient was given an injection of ten cubic centimeters of serum from twenty cubic centimeters of blood on August 26th, after it had stood for twenty-four hours. After the second injection on September 1st of twelve cubic centimeters from twenty-five cubic centimeters of blood, there was slight improvement; however, in the next few days he was just as bad as ever.

Case 5.—J. M., adult. Began to have attacks of hay fever in August, 1911, which came on after an ocean bath. The attack returned in 1912 with asthma, which was cured by a trip to the mountains. He found that taking long walks did him a great deal of good. In 1913 he had an operation on his nose with no relief. And early in August of this year his family physician gave him some serum injections which had no effect on the symptoms.

On August 18th, five cubic centimeters of blood were withdrawn and two cubic centimeters of serum injected. On August 20th, ten cubic centimeters of blood were withdrawn and five cubic centimeters of serum injected, and on August 25th, thirty cubic centimeters were withdrawn, and fifteen cubic centimeters, after standing twenty-four hours, were injected. The injections had no influence whatsoever on his symptoms. The patient has a great deal of nasal obstruction which ought to be corrected.

Case 6.—E. H. O., adult. Has had hay fever for twenty-two years, since he was twelve years old, always accompanied by attacks of asthma. During two years while he was in Europe he was immune. Sea bathing seemed to help him. He had local treatments of adrenalin and caustics. In 1909 a submucous resection was performed, as a result of which he was free of hay fever for that summer, but it reappeared in 1910. He had tried Dunbar's treatment without success.

On August 24th, thirty cubic centimeters of blood were withdrawn and ten cubic centimeters injected, and on August 31st twenty-five cubic centimeters were withdrawn and fifteen cubic centimeters injected with some slight improvement. A further report from the patient states that his hay fever symptoms were as bad as ever.

Case 7.—C. L., adult. Has had hay fever for the past ten years, first appearing at sixteen years of age. He has tried almost every kind of hay fever cure, but got no relief except by going to the mountains. A nasal operation was performed in June, 1913, which cleared the nasal passages to some extent and made breathing easier, but did not relieve the hay fever.

This patient was given five injections, ranging in amount from two cubic centimeters to twelve cubic centimeters. At the time of each injection he was considerably improved for that day, which he seemed to think was due to the relief of the congestion by the withdrawal of the blood. For the first five days after the two injections on August 18th and 20th, he was practically cured, but then he went on an automobile trip, after which his symptoms returned and were as bad as ever. The treatment was of no avail.

Besides these seven cases, five others were treated, the reports of which would be but a repetition of the above. Each was given two or more injections, ranging in amount from five cubic centimeters to fifteen cubic centimeters of serum. In every instance the treatment was a failure.

THE ANAPHYLACTIC PHASES OF HAY FEVER.

There has perhaps been no one disease in which so many remedies have been tried as in hay fever. Individual idiosyncrasies are apparent. One patient will be cured or relieved by adrenalin sprays, another by the ultraviolet rays, another by active bodily elimination, another by one of a dozen quack remedies on the market, another by osteopathy, another by Christian Science, etc. Certain patients have been decidedly relieved by operative procedures upon the nose. There is no doubt that the correcting of nasal deformities and obstructions, and the elimination of various diseased processes in the nose, will alleviate the symptoms for a certain time at least, and it is even productive of cure in a few cases.

Ballenger,³ in his latest work, states that he believes the

primary cause of hay fever in many instances to be an affection of one or more of the sinuses, principally an affection of the ethmoidal cells.

Lloyd, in his book on hay fever, claims that he has cured and relieved eighty per cent of his cases by proper local measures. "It cannot be too distinctly stated that eighty per cent of cases of hay fever and asthma can be cured if the directions which are given are carefully followed." Among those directions are the use of the cautery and the knife.

There is no doubt in the writer's mind that the primary irritation in all cases of hay fever is in the nose, although it must be remembered that the mucous membranes of the eyes, throat, and lungs are also affected. For this reason it is universally considered that hay fever is essentially a nasal condition. It is just as reasonable to suppose that urticaria is a skin condition. Unfortunately, thus far we have been handicapped in our search for the basic cause of hay fever. The writer believes that his premises can be substantiated when he states that hay fever is an anaphylactic condition.

In order to corroborate this opinion, it is necessary for us to study more carefully the various phases of anaphylaxis and serum disease. The first studies along this line were on cases where injections of antitoxin had been given for diphtheria; since that time the various anaphylactic reactions of other irritants have been repeatedly observed. For example: It is known that certain susceptible individuals are anaphylactic to iodoform, and other individuals to other drugs. This was formerly considered hypersusceptibility.

The most important studies within recent years in anaphylactic conditions are those of Anderson and Rosenau, and Geo. H. Weaver.⁴

An interesting case of anaphylaxis is described by Milton,⁵ in which he states that a certain child of ten months, under his observation, was anaphylactic to egg albumen, which gave rise to laryngeal stridor, fever to 103°, an urticarial rash, and a swelling of the tonsils. The symptoms came on within a half hour after feeding the child with egg albumen. Tolerance was found when one drop of egg albumen was added to a quart of water. This child was gradually trained to the use of albumen by diminishing the amount of the dilution day by day.

Another interesting case is reported by Smith,⁶ of buckwheat poisoning. The patient could not tolerate even a small amount of buckwheat.

Experimental anaphylactic reactions have been obtained in animals, principally by the use of various sera from other animals.⁷ One could go on to state innumerable cases of the same sort.

Among other phases of anaphylaxis the following are mentioned by Moschcowitz: (1) A previous susceptibility. (2) An exceedingly minute quantity of toxin is capable of inducing the symptoms. (3) Experimental reactions can be obtained by mouth as well as by injection. (4) The toxin may enter through a mucous membrane. (5) The susceptibility is transferable by heredity. (6) The symptoms are both local and general.

When anaphylaxis is induced experimentally the symptoms noticed are usually as follows (Moschcowitz): Restlessness, coughing, sneezing, swelling of the mucosa of the nose and pharynx, rapid and irregular respiration, and apparent dyspnea. This stage is followed by symptoms of cyanosis, finally respiratory paralysis and convulsions, resulting in death. Such symptoms have been noted in so-called serum disease in human beings.

The similarity between the phenomena of experimental anaphylaxis in animals and serum disease in human beings to asthma, hay fever, and urticaria are very apparent. Serum disease occurs only in susceptible individuals. "The toxic substance enters by the mucous membrane, and is specific for the susceptible individual. That asthma, hay fever, and urticaria are frequently inherited is an observation known to every clinician. Furthermore, the quantity of toxic substances necessary to bring forth symptoms in any one of these maladies is extremely small" (Moschcowitz.)

Another point of similarity between hay fever patients and animals with anaphylaxis may be noted in the fact that in both conditions there is swelling of the mucous membranes of the nose and throat, associated with sneezing and coughing.

We have thus far established the fact that certain symptoms in hay fever may be simulated by experimental work on animals, and that certain phases of serum disease show symptoms analogous to those of hay fever. The relationship between

hay fever and asthma is also significant, in that symptoms of the latter are seen in anaphylacticized animals.

It has also been noted above that a very minute quantity of irritant is necessary in order to bring about certain anaphylactic reactions—i. e., egg albumen, and buckwheat. It is not, therefore, unreasonable to suppose that in order to get reactions in the human being which might be considered anaphylactic, only small doses of the irritant would have to be introduced into the system. It is impossible to state definitely in any given case just how much of a certain irritant will cause a reaction, and it is also impossible to state in just what individuals reactions will occur. Perhaps the reason why very few of these patients respond to the remedies usually applied is that the basic cause of the condition has never been discovered, and that the susceptibility of the individual varies greatly. We do, however, know that there are certain systems which are hypersensitive to specialized agencies, and very often these agencies are those which are never discovered until the individual gets such agency within his system. The best evidence of this fact is the numerous cases of poisoning—i. e., severe urticaria—induced by crab meat and other shell fish. Such a food may be given to one hundred individuals at the same time, and only one individual will be found anaphylactic.

Returning now to the study of hay fever, we must assume the hypothesis, which has been more or less proved, that this disease usually manifests itself by introducing into the system certain toxic substances, usually in the form of minute granules of pollen. That hay fever patients are anaphylactic to pollen and to nothing else, and that normal individuals are not, has been proved by the experiments of Clowes,⁸ in two papers published in 1913. He states that all attempts to induce hay fever symptoms in normal individuals by introducing into their eyes pollen extracts mixed with the serum and nasal secretions of sensitized individuals have failed, and he also proves that the average hay fever patient is extremely sensitive to some substances to which the ordinary individual will not react. "It may be concluded that specific enzymes capable of splitting pollen protein and liberating a toxin factor, or immune bodies capable of combining with a specific body present in the pollen extract to form a toxic combina-

tion, are either not present or not demonstrative in the blood or secretions of hay fever cases." (Clowes.)

The susceptibility of hay fever patients to pollen itself varies greatly, and the writer believes that in certain experiments which he has in view, he will be able to determine with accuracy what the susceptibility of the patient is. As a rule, only minute particles of the pollen are necessary to produce symptoms; so that in experiments such as these, dilutions must be made up in the hundreds of thousands. When once the susceptibility has been ascertained, it will then be necessary to immunize the individual in the same way that other individuals and animals have been desensitized when they have shown special anaphylactic reactions.

It also must be allowed that patients having hay fever inhale vast quantities (comparatively speaking) of pollen from various kinds of plants. Certain patients develop symptoms of hay fever only from one or two kinds of pollen, and in order to effect a cure (of which more will be said later) it is necessary to find out what the individual susceptibility is. For example: Clowes found one woman who was anaphylactic to timothy pollen but not to ragweed.

Moschcowitz draws attention to the fact that the various common diseases in which anaphylactic reactions are present are usually associated with an eosinophilia. "In asthma, hay fever, and urticaria the blood contains an excess of eosinophiles; the secretions of the bronchus and of the nose in asthma and hay fever, respectively, are filled with eosinophile leucocytes. In urticaria also eosinophile cells are abundant in the wheals . . . An eosinophilia, both local and general, is found in nearly all skin lesions (e. g., eczema, prurigo, Dühring's disease, pemphigus, psoriasis, etc.) . . . In connection with the association of many of the manifestations of the exudative diathesis in 'neurotic' individuals, it is significant that Neusser and others have shown that eosinophilia is a common finding in such individuals."

A GENERAL SURVEY OF THE NEW METHODS OF TREATMENT.

In taking up this chapter, the writer does so with the full understanding that the final word has not been said. It seems to him that the most important work that has been done within recent years in the treatment of hay fever has been

undertaken by the laboratory worker and not by the rhinologist. The writer would hesitate to name the thousand and one different remedies that have been advertised within recent years in the treatment of this disease, but he must call attention to the fact that up to the present time even the most enthusiastic workers have had to modify their published assertions as to positive results.

We should leave out of consideration at this time individual remedies and operative measures which have been performed for the cure of hay fever. For although each and every one of these may have an individual value, taken as a whole, no definite success can be reported.

The first important communication suggesting that hay fever did not arise from the irritation of hay, but from the pollen of certain flowering grasses, came from the pen of John Elliotson⁹ in 1830. The disease was recognized in America a few years later, a description appearing in Dunglison's "Practice of Medicine."

Charles Blackley, himself a sufferer from the disease, established definitely during the early seventies, that hay fever was a disease due to pollen. He concluded by experiments with pollen on his eyes and mucous membranes of the upper air tract that pollen did produce catarrhal and asthmatic symptoms, and that the graminaceæ produced the most marked symptoms.

Passive immunization was first begun by Dunbar and his pupil, Praussnitz, who was a hay fever sufferer, early in this century. They brought out a preparation called "pollantin," which can be obtained in the form of a liquid, powder, ointment or pastille.

The preparation is obtained as follows: Horses are selected which show a suitable reaction by the injection of small doses of pollen protein—characterized by a local swelling and urticaria. The selected horses are given increasing doses of the pollen protein at intervals for months, when they can be kept in a sensitized condition. At the height of this sensitiveness (which can be measured) they are bled, the serum is separated, and phenol added. The potency is measured on hay fever patients by ophthalmic reactions. A great many patients react markedly to as small a dose as one-two hun-

dredth milligram. For more definite information one should consult the papers of Dunbar and Praussnitz.

As to the results of Dunbar's serum, one cannot say positively. Certain authors claim that fifty per cent of the patients can be kept free from symptoms, and twenty-five per cent show a partial result. The German Hay Fever Association states that a full success has been observed in about fifty per cent of cases, and a partial success in twenty-five per cent. Some patients claim that pollantin has made them worse. Most of the writer's patients suffering from hay fever have tried Dunbar's serum with absolutely no beneficial result.

Graminol was put on the market soon after Dunbar's serum. Weichhardt worked on the theory that a serum obtained from cattle during the flowering season should contain certain immune bodies. Apparently, from the report of the German Hay Fever Association, the results from this product were as satisfactory as from pollantin.

Active immunization with pollen protein has been attempted with fairly successful results in the past few years. Most of this research work has been done by laboratory workers, more from an experimental point of view than from a clinical or practical one.

Clowes, Lowdermilk, and Koessler have all done work in this line, and it will be exceedingly interesting to detail some of their experiments.

Clowes prepared an aqueous solution of the pollen of ragweed by precipitating the pollen with acetone and extracting with water. He treated eight cases by giving injections of this solution very much diluted, so that the injection varied from one cubic centimeter of 1/5,000,000 dilution to one cubic centimeter of 1/500,000 dilution. He claims that there was a marked alleviation of the symptoms, and states that he believes that the failure of the usual treatment is due to giving too strong injections.

During the year 1913 Lowdermilk¹⁰ treated nineteen cases of hay fever with successful results in sixteen. The toxin was prepared as follows: One gram of mixed pollen of different varieties of plants found in that locality was mixed with ten grams of sterilized fine sea sand in a sterile mortar: one hundred cubic centimeters of 1 physiologic salt solution, in

which was a small amount of carbolic acid, was measured out and the pollen moistened with a portion of it. The mass was then ground for several hours, with the addition of portions of salt solution, until the sand was reduced to an impalpable powder. The remainder of the salt solution was then added, the whole transferred to a sterile flask and allowed to stand at room temperature for twenty-four hours with frequent shaking. The fluid was then pipetted off, centrifuged and sealed in glass ampules, each containing one cubic centimeter. The unit is the quantity of toxin extracted from one microgram of pollen. As prepared, each cubic centimeter of stock solution contained ten thousand units. From the stock solution two serial decimal dilutions were made, containing one thousand and one hundred units to the cubic centimeter. By using a tuberculin syringe it was possible to administer doses containing any desired number of units. Lowdermilk was unable to determine the proper dosage in advance by the usual methods, and relied entirely upon the nature and extent of the reactions obtained. In certain cases, where pus seemed to be present in the nose, autogenous vaccines were given.

Koessler¹¹ gives in most illuminating detail the most salient points of the work on hay fever up to date. He reviews the work of active immunization by Curtis, Wagner, Ingals, Noon and Freeman, Clowes and others; and in 1910 began active immunization of hay fever patients. He has treated forty-one cases by his method during four years. Five had spring catarrh, thirty-six had autumnal catarrh. Seventeen had prophylactic treatment, and nineteen were treated during the attack. Four have been completely free from symptoms. Of the four, three had treatment over two seasons and one over three. Twenty-nine were very much improved, both subjectively and objectively, while eight cases showed little benefit. Twenty-three of the forty-one patients had asthma. Of these, sixteen were much benefited, six having no asthma.

Koessler's preparation was made from ragweed, and a saline extract was obtained as follows:

One-tenth gram (one centigram) of pollen is broken up as finely as possible in an agate mortar, and gradually ten cubic centimeters of an eight and one-half per cent salt solution, ten times as strong as a physiologic salt solution, is added drop by drop. This saline suspension is shaken for two hours and

then left in the incubator at 37° centigrade for sixteen hours. Then the extract is again shaken for two hours, centrifuged, and the supernatant fluid separated with a pipette from the undissolved residue. The supernatant fluid, which is a dilution of 1/1000, is diluted ten times with sterile distilled water plus .25 per cent phenol, which makes the salt solution a physiologic one, and the dilution 1/100,000. From this dilution all others are prepared. This dilution and all lower ones are unstable and deteriorate by progressive proteolysis into a toxic product within eight to ten days. The concentrated pollen solution in eight and one-half per cent saline is more stable, and can be kept on ice for three weeks.

An alcoholic extract can be made by precipitating a saline extract in ten times its volume of ninety-five per cent alcohol.

The minimum toxic dose was determined by dropping solutions onto the conjunctiva of a hay fever patient until a marked hyperemia was produced. This varied between one-twentieth cubic centimeter of a 1/500,000 dilution to a one-twentieth cubic centimeter of a 1/10,000 dilution. Persons who are not susceptible do not show any reaction, even when large amounts are used. The initial immunizing dose used by Koessler was one-half the amount needed to give an ophthalmic reaction. The injections are given subcutaneously, and are repeated at intervals of four to ten days, depending upon the amount of immunization, as determined by the ophthalmic reaction. It is best to give prophylactic treatment, starting in May, but Koessler has been encouraged by the results obtained during the actual hay fever attack.

The writer is beginning a new series of experiments along the lines here suggested, which he hopes will add still more to our knowledge of this subject. Koessler's work is very encouraging and deserves the recognition of a great scientific work. The writer feels that he can best conclude by repeating Koessler's last words:

"I wish to express emphatically a word of caution. It will not be long before the commercial manufacturers of vaccines see 'the great advantage and benefit' of this treatment. Hay fever vaccines will be praised and advertised and put up so attractively that their use will become universal, and soon universally discredited. For the pollen extract is not stable, especially not the higher dilutions. By progressing proteoly-

sis, after three to four weeks, it acquires marked toxic properties which lead to severe reactions. The solutions must, therefore, be freshly prepared every eight to ten days, if these reactions are to be avoided. Whatever the method of active immunization, whatever the dosage and technic, the one sound basis that must underlie all these endeavors is that the material to be injected must be not only sterile, but constantly of uniform potency if used in the same dilution. No extract of pollen can comply with this demand if it is older than three weeks."

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XX

ACCIDENTAL PNEUMOTHORAX DURING TRACHEOTOMY, WITH REPORT OF A CASE.*

BY SAMUEL IGLAUER, M. D.,

CINCINNATI.

In the literature on tracheotomy one seldom finds any mention of the occurrence of pneumothorax as a complication of the operation. The fact that this complication may occur was rather forcibly called to my attention in a case recently under my care.

The patient, a male child, aged twenty-three months, was brought to my office by Dr. M. Behrman, on Friday morning, January 8, 1915. The doctor stated that about two o'clock of the same morning he had been hurriedly summoned to relieve the child of a severe choking spell which subsided somewhat after he arrived and had applied adrenalin to the larynx.

The family physician and Dr. Behrman had been in attendance on the child since the previous Wednesday, at which time, while at breakfast, the baby had had a sudden choking spell while eating a piece of rabbit. The child's mother had attempted to dislodge the foreign body (supposed) with her finger. At the time of the physician's first examination nothing abnormal was observed in the throat except a slight trace of blood.

After the initial attack the child became hoarse but not croupy, while wheezing respiration without cough was noted at times. On the following day the wheezing respiration became more labored. No fever was noted during the illness, and the physical examination of the chest had revealed only slight variations from the normal.

When I saw the child it was suffering with severe inspiratory dyspnea, increasing paroxysmally at times. There was marked inspiratory retraction of the lower thorax. Cyanosis at times became extreme.

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A direct examination of the larynx failed to show a foreign body or membrane. Owing to the urgency of the case the chest was not examined. The diagnosis of foreign body in the air passages was made from the history and symptoms.

Operation.—An incision for a low tracheotomy was begun without an anesthetic. The isthmus of the thyroid gland was exposed at the upper margin of the wound, and the apex of the thymus gland presented at the lower angle. Before the trachea had been opened I noticed fairly large bubbles of air collecting at the lower angle of the wound. This continued for a short time. The air seemed to be drawn into the wound and then bubbled out. Throughout the operation the child struggled continuously, so that I found it necessary to summon my neighbor, Dr. Mithoefer, to aid in the operation. Ether was then given and the tracheotomy easily completed. Immediately after the tracheotomy tube was inserted the child collapsed and became almost pulseless, with extreme pallor and cold extremities. Camphor was injected hypodermically and the patient revived.

Subsequent History.—About one hour after the operation Dr. Lange made a roentgenogram of the chest. This failed to reveal a foreign body, but plainly showed a pneumothorax of the right side, with almost complete collapse of the lung and a transposition of the mediastinum toward the left.

The child was taken to the hospital. Physical examination confirmed the X-ray findings. A slight emphysema at the root of the neck disappeared when the lowermost stitch was removed.

Several days after the introduction of the tracheotomy tube I thought I could hear air bubbling in and out alongside the tube, but of this I was not certain. The right side of the chest was strapped for a few days, to prevent the inspiration of secretions into the pleural sac.

Signs of diffuse bronchitis developed in the left lung. On the third day after operation a small bronchoscopic tube was introduced, but no foreign body was found. A catheter was also passed from the wound through larynx into the pharynx without dislodging any foreign body. During the first few days after the operation the temperature ranged about 102° to 103°; the pulse about one hundred and forty to one hundred and sixty; and the respiration ranged about sixty. Oxygen and stimulants were administered at frequent intervals.

From this time on recovery was more rapid. There was some difficulty in dispensing with the tube, which, however, was finally removed on the twentieth day, at which time the voice had fully returned. The lung had reexpanded by the twenty-seventh day, when the child left the hospital.

Comment.—The points of especial interest in this case were the accumulation of bubbles of air in the lower angle of the wound before the trachea had been opened, the sudden collapse immediately after the operation, the early detection of the pneumothorax by the roentgenogram, and the rapid respirations during the first few days after operation. While the diagnosis of a foreign body seemed justifiable, the case was probably one of acute subglottic laryngitis.

EXPLANATION OF THE OCCURRENCE OF PNEUMOTHORAX IN CONNECTION WITH TRACHEOTOMY.

The association of pneumothorax in connection with tracheotomy has been the subject of research by F. H. Champney.¹ His experiments were made on the bodies of stillborn children. A tracheotomy was performed and a cannula was tied into the trachea. This cannula was connected by tubing with a manometer. The movements of artificial respiration were then performed. Mediastinal emphysema ensued in seven out of twenty-one of the bodies, and pneumothorax occurred in five of these seven cases.

"Pneumothorax never occurred without emphysema, and was probably a later sequel of the emphysema. . . . Air was observed to escape from the mediastinum into the pleural sac. . . . It appeared then that the air traveled from the tracheotomy wound into the mediastinum, which in some cases it ruptured, producing pneumothorax."

Champney's explanation¹ may be summarized as follows: If the entrance of air into the trachea be interfered with during forcible inspiratory efforts, and if at the same time the deep cervical fascia be opened as a preliminary to tracheotomy, then a potential passage into the thorax becomes established, and it is through this passage that the air penetrates the mediastinum. If the pressure is equalized, it goes no further; but if the pressure is not equalized, it tends to burst the mediastinum and distend the pleural sac.

Mediastinal emphysema is due to diminished pressure in

the thorax during inspiration, in contradistinction to the common form of emphysema of the neck after tracheotomy, which is purely of expiratory origin.

In addition to Champney's explanation, the occurrence of pneumothorax might also be accounted for by the accidental wounding of the pleura during tracheotomy; but this contingency is very remote, is anatomically almost impossible, and would doubtless be associated with severe hemorrhage from some of the great vessels at the root of the neck.

The chief rules laid down by Champney (op. cit.) for the avoidance of emphysema and pneumothorax are: First, to avoid low incisions in the deep cervical fascia; and second, to refrain from elevating the deep fascia before the trachea is opened. As illustrated in the clinical case recorded above, a struggling patient and a low tracheotomy seem to be predisposing factors.

As to the frequency of pneumothorax complicating tracheotomy, Champney² was able to find a postmortem record of six cases. Mediastinal emphysema was mentioned much more frequently. It is a fact worthy of note, however, that in a single London hospital careful postmortem examinations, under water, revealed more cases of this nature in the two years following the publication of Champney's paper than had been recorded in the same hospital in the twenty-one preceding years. Leiner³ records a series of twelve cases of tracheotomy in children, in nine of which mediastinal emphysema ensued. Crepitation, synchronous with the heart action, was the chief symptom observed.

Owing to the circumstances under which tracheotomy is usually performed, the occurrence of mediastinal emphysema and pneumothorax may easily be overlooked, but close observation during the operation, as well as careful physical and radiographic examination after tracheotomy, will doubtless reveal the presence of one or both of these complications in a considerable number of cases pursuing an abnormal course.

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XXI.

A SIMPLE MANOMETRIC APPARATUS FOR MEASURING EUSTACHIAN TUBAL PATENCY AND TYMPANIC OSSICULAR MOBILITY.*

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In the diagnosis, prognosis and treatment of the various degrees of tubal stenosis and ossicular immobility, some method of measuring is necessary.

This apparatus, arranged for measuring eustachian tubal patency, consists of the following parts: A spring manometer, gauging from 0 to 260 mm. of mercury pressure, attached by tubing to a T connecting tube; a rubber caudery inflation bag attached to another end of the T; attached to the third end of the T is a rubber tube connected with a stopcock, and from this another tube ending in a nasal acorn tip.

The tube leading from the stopcock to the nasal tip must be of inelastic, rather heavy rubber, so that the air pressure will not be lowered by the expansibility of its walls. If a light rubber, instead of an inelastic rubber, tubing is used, the difference in reading before and after opening the stopcock amounts to twice as much with the higher pressures, such as 200 mm., when forcing air into cavities of from one-half to one and one-half ounce capacities. Thus with the one-half ounce chamber connected, instead of a difference of 7.50 mm., there will be a difference of 15 mm. of pressure.

The acorn tip should be about 2 cm. in diameter, so that it will not be forced into the nostril on pressure.

The method followed is to close the stopcock, raise the air pressure to 10 mm. of mercury with the inflation bulb, close the epipharynx and the other nostril in the usual manner of politzerization, then at the proper moment release the air into the nostril by turning the stopcock. If this air pressure is

*Read before the New York Academy of Medicine, Section on Otology, January 8, 1915.

insufficient to open a tube and enter a tympanum, it is raised 5 cmm. at a time until the required amount has been found for each tube.

If one method fails to close the epipharynx as indicated by immobile *alæ nasi* on inflation, e. g., on deglutition of water, then other methods must be tried seriatim, such as saying KKK, puffing out the cheeks, etc.

To avoid errors due to the walls of the tube being adherent or plugged with mucus, the tubes can be gently inflated before making the test, or the tests can be repeated until the exact amount of pressure required is determined.

In making the tests with some patients, it is necessary to use the diagnostic auscultation tube, on account of their inability to indicate when the air enters the tympanum.

In those instances where politzerization fails, the inflation by catheter can be used, substituting a catheter tip for the nasal tip. With the catheter, however, more elements of error are introduced, so that longer tests are needed to decide the mensuration.

This test is not intended to express the absolute air pressure in the epipharynx, as the release of the air into the terminal tube, the nasal chambers and epipharynx, lowers somewhat the pressure of air at the pharyngeal ostium. However, as the capacity of the cauter bulb chamber and tubing to the stopcock is over ten and one-half ounces, and that of the terminal tube (three drams), nasal chambers and epipharynx (when the nostrils are compressed and the soft palate raised) varies from less than half an ounce to one and a half ounces, there is not a great difference between the pressure in the bulb and that in the epipharynx.

The chart was made with this apparatus and is presented to show the approximate corrections, if it is desired to know the epipharyngeal pressure. Note that until 50 cmm. pressure is reached there are no corrections to be made.

Arranged for the Gellé test, a six ounce Politzer bag is substituted for the cauter bulb, as in this instance sudden pressures and sudden releases from pressure are required.

In place of the nasal tip, an olivary aural tip of correct size, covered with thin rubber tubing, is placed on the terminal tube. It is necessary to have four or more sizes of aural tips,

and the olivary form, covered with rubber, causes the least discomfort from the firm pressure required.

The method of procedure is to insert the aural tip firmly in the external auditory meatus so that there is no air leakage on inflation, start the tuning fork and place over the mastoid antrum, care being used not to have the fork sound too loudly (so as to eliminate the other ear as much as possible), then raise the air pressure suddenly to 10 mm. of mercury, then lower it to zero, repeating this several times. Then try 15 mm., and so on up, until you discover the pressure needed to alter the audition for the fork tones.

Unfortunately, the test is of value only in the earliest stages of ossicular immobility.

Table showing changed air pressure and the difference in air pressure, when air is released from the apparatus into chambers of one-half ounce, one ounce and one and one-half ounce capacities:

Mercury in Cu. mm. of Air Pressure	$\frac{1}{2}$ ounce		1 ounce		$1\frac{1}{2}$ ounces	
	After Release	Difference	After Release	Difference	After Release	Difference
10.....	10.	.00+	10.	.00+	10.	.00+
20.....	20.	.00+	20.	.00+	20.	.00+
30.....	30.	.00+	30.	.00+	30.	.00+
40.....	40.	.00+	40.	.00+	40.	.00+
50.....	49.38	.62	49.38	.62	49.38	.62
60.....	58.75	1.25	58.75	1.25	58.75	1.25
70.....	67.50	2.50	67.50	2.50	67.50	2.50
80.....	77.25	2.75	76.88	3.12	76.25	3.75
90.....	87.00	3.00	85.62	4.38	85.00	5.00
100.....	96.25	3.75	95.00	5.00	93.75	6.25
110.....	105.75	4.25	104.50	5.50	103.75	6.25
120.....	115.00	5.00	113.75	6.25	112.50	7.50
130.....	125.00	5.00	122.50	7.50	121.25	8.75
140.....	135.00	5.00	132.50	7.50	131.25	8.75
150.....	145.00	5.00	142.50	7.50	140.63	9.37
160.....	154.25	5.75	152.50	7.50	150.00	10.00
170.....	163.75	6.25	160.00	10.00	158.00	12.00
180.....	173.25	6.75	170.00	10.00	167.50	12.50
190.....	182.50	7.50	178.75	11.25	176.25	13.75
200.....	192.50	7.50	188.75	11.25	185.00	15.00

Note.—These figures are only approximately correct.

XXII.

ACCIDENTAL INJURIES OF THE SIGMOID SINUS INFLECTED IN SIMPLE MASTOIDECTOMY.*

BY WILLIAM A. SCRUTON, M. D.,

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In a review, which is believed to be complete, of the reported cases of septic thrombosis of the sigmoid sinus, I have failed to notice one in which penetrating wound during mastoidectomy is mentioned as an etiologic factor. Examining ten textbooks on otology, I found the danger of septic thrombosis following injury of the sinus mentioned in one only, and in that the danger is considered slight.

During my resident hospital service I am certain that I have seen ten or twelve accidental penetrations of the sinus, and having the cases under my observation subsequently, know that none of them developed a septic thrombosis. In no instance were special precautions taken to guard against sepsis at the site of the rupture. The hemorrhage was controlled by a plug of iodoform gauze and the entire cavity packed tightly—generally with iodoform gauze, in a few cases with plain gauze.

Cases closely allied to the subject have been reported. Bondy¹ cites a case of Marum, in which puncture of the sinus with a diagnostic needle resulted nine days later in operation for septic thrombosis. Green² reports a case in which the patient having died of other complications, autopsy demonstrated a sterile clot at the site of a diagnostic needle puncture. John R. Page³ reports "a case of profuse hemorrhage from the middle ear immediately after myringotomy in an infant eleven months of age, followed by signs of jugular thrombosis with recovery after resection."

My personal experience with accidental injury of the sigmoid sinus has been gained entirely within the past year. Con-

*Read before the New York Academy of Medicine, Section on Otology, January 8, 1915.

cise histories of the cases, with temperature charts and conclusions, are appended.

Case 1.—E. K., age fourteen years, whose occupation is student in public school. His present illness occurred during the past month while recovering from a severe attack of coryza. He complained of fullness and pain in the left ear, which was relieved by application of hot salt in a bag. Within two weeks discharge appeared and pain at times became severe. The chief complaint is discharge from left ear, with swelling and tenderness over the corresponding mastoid process. On examination the left ear shows profuse purulent discharge and a thickened canal; the drum cannot be inspected. There is edema in front, behind and below the auricle. Acute mastoid tenderness is demonstrated. The right ear proves negative. He appears sickly and rather under weight; has hypertrophied tonsils and a moderately large adenoid, with a mucopurulent discharge from the nose and nasopharynx. His heart, lungs and abdomen are negative. The laboratory report of the urine analysis is negative. The temperature registers 100.5° F., pulse 110, respiration 22.

A simple mastoidectomy was performed January 6, 1913. The usual postauricular curved incision was made, with a supplementary straight incision backward. The exposed cortex was moderately thick, discolored and soft over the antrum. An opening was made in cortex at the softened area with a curette and extended with a rongeur. The mastoid cavity was found to be a lake of pus. When the pus was removed, more was seen discharging from a necrotic aperture in the bone over the knee of the sinus. The inner plate was removed with curette and rongeur, exposing an extensive epidural and perisinus abscess. In extending the exposure to healthy dura and sinus, the sinus was nicked with a rongeur at the junction of the emissary. At this point the sinus appeared healthy. Unhealthy vessel extended from just behind the knee down to the turn under the posterior wall of the external auditory canal; it was bathed in pus, the wall was discolored and apparently thickened.

Hemorrhage from the ruptured vessel was controlled by a plug of iodoform gauze. The wound was packed with iodoform gauze after flushing with hot normal saline solution. The skin incisions were not closed.

On January 7th the laboratory reported the following result of a blood examination: Leucocytes, 16,000; lymphocytes, twenty-five per cent; polynuclear neutrophiles, seventy-five per cent. A culture of the pus was also reported, showing an uncontaminated growth of staphylococcus.

The third day after operation the temperature registered 102° F.; and from this date (January 10th) to the 17th the temperature chart shows increasingly steep peaks characteristic of septic thrombosis. There were no chills recorded.

Jugular resection and exploration of the sinus was undertaken on January 17th. An incision was made in the neck along the anterior border of the sternocleidomastoid muscle. With blunt scissors dissection the jugular vein was quickly exposed; it was found to be collapsed almost down to the junction of the facial vein. After resection the neck wound was partially closed below and packed with iodoform gauze. The sinus exposed in the mastoid wound was now compressed at the extreme upper and lower limits with plugs of iodoform gauze. On incising the vessel wall between the two compression points, a hemorrhagic clot was demonstrated. This was turned out and the entire vessel wall then cut away flush with the dura. When pressure was relieved at either plug at the extremes of the sinus exposure, free hemorrhage resulted. Finally, the mastoid cavity was flushed with hot normal saline solution and packed with iodoform gauze.

The appended temperature chart shows the subsequent history of this case was a tedious combat with pyemia, terminating in death May 29th. Abscesses were opened as they appeared; they were situated in both sides of the neck, the shoulders and the back. The left knee became swollen and red, but did not progress to abscess formation.

The medical treatment of this case is of interest. Hiss leucocyte extract was given for ten days in January (twenty cubic centimeters subcutaneously twice daily). This treatment was repeated in May for a period of ten days. In February an autogenous vaccine (staphylococcus pyogenes aureus) was employed. Despite these measures, with a free use of stimulating and supporting treatment, aided by a good appetite and excellent digestion, the patient was unable to resist the pyemic invasion.

Case 2.—A. C., female, age thirty-two years; occupation,

housewife. The present illness began eight days ago, while recovering from a sore throat. The onset was marked by sudden severe pain in her left ear. A hot water bottle was applied, and within a few hours discharge appeared; the pain then subsided. Her chief complaint on admission to the hospital was of sharp shooting pains in left side of the head, radiating from the ear. In addition, she complained of cough, malaise, anorexia and constipation.

Examination of the left ear showed a moderate discharge of thick pus, also mastoid edema and tenderness. The right ear was found normal on examination. When inspecting the throat the tonsils were seen to be diseased and the mucous membrane generally congested. Auscultation of the chest revealed the presence of moist rales at the apices of both lungs; the heart proved normal. Examination of the abdomen showed nothing abnormal. The laboratory returned a negative report on the urine; also a negative report on the sputum. A microscopic examination of the pus from ear showed diplo- and streptococci.

The patient did not think there had been any loss of weight during the past year; however, she was frail appearing and suggestive of tuberculosis. She had cough for some months, and frequent attacks of sore throat. Her temperature on admission to the hospital was 98.8° F., pulse 94, and respiration 20.

On February 19, 1913, a simple mastoidectomy was performed. The usual incision was made over the mastoid, following the curve of the auricle, and the periosteum elevated and retracted; this exposed thin discolored cortex which was soft at the tip. A supplementary straight incision backward was now made, in order to gain more working space. The mastoid was opened at the tip by means of a rongeur. Free pus was present in the tip cells and the antrum. The sinus ran almost directly under the cortex, and in close relation with the posterior wall of the external auditory canal. While probing over the lower part of the sinus with a small Spratt curette, endeavoring to determine if the inner plate was intact, the sinus wall was accidentally penetrated. On account of the close relation of the sinus to the posterior wall of the external auditory canal, considerable difficulty was experienced in removing the cells between them—especially after the injury to

the vessel. The inner plate covering the entire sinus in the mastoid excavation was removed. In appearance the sinus was normal. The wound was flushed with hot normal saline solution and packed with iodoform gauze. The skin incisions were not sutured.

This patient after recovery from the anesthesia (chloroform) showed facial paralysis. No twitching of the face having been noted during the operation, it seemed probable that the nerve had not been severed. Complete recovery from the paralysis took place in about four months, under treatment with massage and the galvanic current.

The temperature following operation showed the characteristics of sepsis in the sinus. The peaks of the chart were higher each succeeding day until the 25th of February, when the peak recorded was 104.5° F.

From a blood examination February 26th, the laboratory reported leucocytes, 15,000; polynuclear neutrophiles, seventy-five per cent; lymphocytes, twenty-five per cent.

The wounding of the sinus at operation, the subsequent temperature with chilly sensations, and the leucocytosis led us to decide upon resection of the jugular vein and exploration of the sinus.

The operation was performed February 26th. The usual incision along the anterior border of the sternocleidomastoid muscle was made, and the jugular vein exposed by blunt scissors dissection. The vessel was apparently normal in appearance. In clamping the lower end of the vein the assistant accidentally included the vagus. Although this was immediately released, the patient afterward had a paralysis of the left vocal cord. The normal voice was recovered in about three months. The sinus was compressed above, well back of the knee and below, close to the bulb; then opened between the two compression points. A small clot was found, and free hemorrhage occurred on releasing the pressure plugs at either end of the vessel. The wall of the sinus was cut away flush with the dura. Both neck and mastoid wounds were left wide open and packed with iodoform gauze.

The subsequent temperature, as indicated on the chart, showed three high peaks in a lysis extending over eight days; thereafter recovery was uneventful.

Case 3.—B. C., male, age sixty-four years; occupation, a

sail maker. His present illness began ten days ago, when, after unusual exposure, he complained of fullness in the left ear with occasional slight darting pain. A profuse discharge appeared on the fourth day, and on the fifth day his friends noticed that he did not hear well; however, he made no complaint himself of deafness or of any unusual sensation in the right ear. On the tenth day of his illness he was admitted to the hospital. (March 12, 1913). His chief complaint at this time was bilateral deafness and discharge from the left ear. An examination of the left ear showed a profuse discharge, nonodorous, coming through a small central perforation. There was no mastoid tenderness. The hearing was found to be one foot for loud spoken voice. Examining the right ear the drum was found bulging and about to rupture centrally. The hearing on this side was reduced to within a foot for loud spoken voice. No nystagmus was noted, and he gave no history of vertigo or vomiting. He was a phlegmatic individual with a normal chest and abdomen. The laboratory reported a trace of albumin and a few hyalin and granular casts in his urine. A culture of the pus from ears was reported streptococcus mucosus. On admission to the hospital his temperature was 102.8° F., pulse 100, respiration 26.

Myringotomy was performed on both ears, and orders given to irrigate with hot normal saline solution every two hours. The myringotomy improved the hearing markedly in the right ear, but there was no change in the left. Within a few days the discharge increased from the right side, and the hearing was again reduced to about a foot for loud spoken voice.

From March 12th to 22d the temperature ranged from normal in the morning to approximately 101° F. in the evening. There were no chills noted, and the patient made no complaint of chilly sensations when questioned.

Mastoid tenderness was not present at any time. The temperature chart, increasing discharge, and the character of the infection determined operation. Consent to operate was finally obtained on March 22d.

The operation was a simple mastoidectomy on the right and left ears. On both sides the usual postauricular curved incision was made, exposing thick, hard and normal appearing cortex. The sinuses were deeply situated well behind the pos-

terior wall of the external auditory canal on either side. Free pus was found in both mastoid cavities. It was decided to investigate the appearance of both sinuses. While uncovering the left sinus it was accidentally pinched between the blade of the rongeur and the inner plate of bone and ruptured. The resulting hemorrhage was easily controlled with a plug of iodoform gauze. The macroscopic appearance of the vessel on either side was quite normal. The mastoid excavations were flushed with hot normal saline solution. Packed the left wound with iodoform gauze; the right with plain gauze. Both wounds were unsutured.

On March 23d the temperature reached 106° F. and fluctuated between 104° F. and 106° F. for the next three days, finally reaching 108° F. before death on the fifth day.

The laboratory reported on blood examination March 18th: Leucocytes, 7,000; lymphocytes, twenty-five per cent; polynuclear neutrophiles, seventy-five per cent. On March 24th the report was: Leucocytes, 12,000; lymphocytes, eighteen per cent; polynuclear neutrophiles eighty-two per cent, and culture of blood showed a growth of a hemolytic streptococcus.

Case 4.—E. B., colored, female, age sixty years; occupation, a janitress. Her present illness began four weeks ago during an attack of acute coryza. The patient suffered a severe pain in the right ear. The pain subsided in a short time and a profuse discharge appeared. On admission to the hospital her chief complaint was pain over the right side of the head and discharge from the right ear. She also complained of malaise, anorexia and constipation. Examination of the right ear showed a profuse discharge associated with a very exquisite mastoid tenderness. The left ear was also examined and was found apparently normal. The patient was a well nourished, healthy appearing woman. Examination of the chest and abdomen gave a negative result. The laboratory reported her urine as negative. The pus from the ear was reported to contain staphylococci. Her temperature at the time of admission was 100° F., pulse 96, and respiration 22.

A simple mastoidectomy was performed on October 13, 1913. An incision was made over the mastoid process, following the curve of the auricle; this was enlarged by a supplementary straight incision backward. When the periosteum was retracted, the exposed bone appeared soft and discolored.

Removing the thick cortex with rongeur, a large area of cellular zygomatic region was exposed containing pus. There was free pus in the antrum and in some of the cells at the tip. The sinus was found deeply seated, well back of the posterior wall of the external auditory canal. It was exposed by means of the curette and rongeur, and was normal in appearance. In removing the inner plate a small sliver of bone accidentally penetrated the sinus. The resulting hemorrhage was checked by a plug of iodoform gauze.

In my previous cases no procedure, other than hot normal saline irrigation, was directed toward cleansing of the mastoid cavity following penetration of the sinus. In the present case the following precautions were instituted: The wound was treated with hydrogen peroxid, then flushed with hot normal saline; this was followed by alcohol ninety-five per cent and the hot normal saline flushing repeated. The plug was removed from the sinus and the blood allowed to flow a few seconds. A fresh iodoform gauze plug was applied, the blood flushed away with hot normal saline, and the wound packed snugly (not tightly) with folded iodoform gauze in strips; one end of each strip being brought outside of the wound. The skin incision was partially sutured.

From the second to the fourteenth day following operation, a full dressing was done daily in this fashion: The packing was moistened with hydrogen peroxid and gently removed, to avoid disturbing the plug over the sinus rupture; the mastoid cavity was then wiped dry and the folded iodoform packing replaced. On the sixth day, after the packing had been removed, hydrogen peroxid was applied directly to the plug on the sinus wall and a little manipulation begun. No force was exerted. This procedure was carried out at each dressing thereafter, and on the fourteenth day the iodoform plug had practically freed itself.

Subsequent to the operation the temperature reached a fraction above 100° F. on the third day. It then returned to normal and remained approximately so until the patient was discharged from the ward.

CONCLUSION.

We are taught that the formation of clot within a blood vessel, which is a stage in one of the repair processes of

nature, occurs only when the latent function of the intima is stimulated to activity and a fibrin ferment liberated. The stimulation may be the result of toxic, bactericidal, inflammatory or traumatic irritation.

Accidental injury of the wall of the sigmoid sinus, primarily would appear to be of no great consequence. It becomes of serious importance, however, when infection gains entrance to the protective clot. The clot itself then undergoes dissolution, throwing toxins and living bacteria into the circulation.

It is probable that most cases of injury of the sinus wall do not result in septic thrombosis, because the sudden forceful outrush of blood carries with it the contamination introduced by the offending instrument and also tends to wash away septic material from the vicinity of the injury.

It would seem desirable when injury of the sinus occurs to undertake a more than usual antiseptic treatment of the mastoid cavity, as an additional precaution against infection of the protective clot.

In case number one the sinus being apparently involved in inflammatory process throughout the entire thickness of the wall, septic thrombosis might possibly have resulted even without the injury having been inflicted. The sinus in case number two was normal in appearance. In case number three, following the penetration of a normal appearing sinus, there almost immediately resulted an overwhelming bacteremia (*streptococcus mucosus*). This tends to show that the character of the invading organism has an important bearing on the prognosis.

Three of the four cases in this series developed septic thrombosis, of which two resulted in death.

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XXIII.

ETHMOIDAL EXENTERATION.*

BY CHARLES M. ROBERTSON, M. D.,

CHICAGO.

You are all acquainted with the Mosher operation for cases, not of acute ethmoiditis of a cell, but of a condition involving the entire ethmoidal chain. This paper will suppose that local treatment has been done in the nose, and that perhaps a cell or more have already been opened, with failure to relieve the case. Exenteration of the ethmoids is an operation to be performed in the type of cases in which local treatment and local operations have failed. There are certain anatomic defects in the nostril that are prone to produce ethmoiditis, and prominently among these I desire to mention one particular type. I do not know whether all of you have seen it or not, but I think that this type of ethmoiditis has not been dealt with sufficiently. That is, a type in which the septum is deflected toward the side affected, either by a bowed septum or a thickening of the upper plate—that is, of the ethmoidal vertical plate. These cases, as the books speak of them, are those in which reference is made to bowing of the septum, and also of a piling up of tissue around the junction of the quadrilateral cartilage and the bony walls. Many cases have come to my notice of deflections of the septum, where there have been submucous resections made, and these submucous resections have been limited to the quadrangular cartilage and the lower part of the bony wall of the septum. Nearly all of these cases have a thickening of the bony vertical plate of the vomer as high up as the cribriform plate, and these are cases in which, if the case is operated upon (by submucous resection), resection should be made to the cribriform plate.

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There is another type in which the cells in the middle ethmoid bone are very large. There is a cystic degeneration of the middle turbinate. These ethmoid cells are not present originally, but are produced by some inflammatory condition in the tissues and the continuation of the ethmoid cell into the middle turbinate body. This probably is a seromucous inflammation, and the cells are produced by an expansion of a cell. Some of these cases go on to suppuration, and some never suppurate. I have seen several cases in which the turbinate appeared practically normal, only that the cells in the turbinate had produced an enlargement of the turbinate, producing an enlargement sufficient to push the bony wall of the septum over and make it deflect upon the other side. These are cases in which, if the turbinate be resected, the resiliency of the wall of the septum will spring it back into place. I have seen cases of this type operated on by submucous resections and the turbinate left in place. That is the wrong thing to do.

In exenteration of the ethmoid cells, we can exenterate part or all of them.

In a paper on this subject that was read by the speaker three or four years ago, at the Chicago Medical Society, I brought out an operation which I desire to bring before your notice, in which the ethmoid cells are attacked through the bulla ethmoidalis. The operation is as follows: The ethmoid cells are exenterated and the middle turbinate saved. The operation is done after the style of Mosher, except that Mosher goes in in front, on the inner surface of the middle turbinate, at its anterior end, and breaks through into the front or ethmoidal cells. Then he tears back through the ethmoid cells and tears away the middle ethmoid bone, destroying the superior and middle turbinate bodies in one piece. With the operation I desire to call your attention to, the entrance through the bulla ethmoidalis allows you to get the lower middle ethmoid cells, and from this, if you follow back, you exenterate the posterior ethmoid cells quite easily; then, if you bring your curette forward (with an angular curette) you can get pretty well forward towards the anterior ethmoid cells.

In these cases, after the exenteration of the ethmoid is done, the middle turbinate body is fractured at the top and pushed over into the space which was ethmoid cell.

I have done several of these cases, and have watched them to see if I had an atrophy of the lateral mass of ethmoid, and to see what effect it had on the nose, regarding ventilation, and it appeals to me of sufficient importance to bring it to your attention—that the ethmoidal cells can in this way be absolutely obliterated and the turbinate saved, which is a point that is very important in the physiology of a nostril.

XXIV.

THE USE OF PITUITARY EXTRACT AS A COAGULANT IN SURGERY OF THE NOSE AND THROAT.*

BY HARRY KAHN, M. D., AND L. E. GORDON, M. D.,

CHICAGO.

The rhinologist has long sought a harmless, efficient drug that will control the hemorrhage during and following operations on the nose and throat. Calcium salts have been used with indifferent success. Blood serums, either freshly prepared from the human or from the rabbit or the horse, have reliable results, but have the disadvantage that they may produce anaphylaxis. Adrenalin only produces its results on the mucous membranes, and then only when directly applied.

Citelli¹ reported to the Seventeenth International Medical Congress, in London, August, 1913, on the use of pituitary extract for the control of hemorrhage following turbinectomy, sinus operations, and tonsillectomy. Following his suggestion, we have used the drug, and with universally good results. The blood loss during and after turbinectomies was slight; in fact, the operation itself was almost bloodless. The hemorrhage following curettements of the ethmoids, also. The blood loss following the tonsil operation was greatly reduced. The snare, Sluder and Beck methods were used for the tonsil enucleation. Not a single untoward result have we had in upwards of over one hundred cases operated on, which could be attributed to the drug.

The action of this drug as a blood-stilling agent did not seem to us to be wholly explained by the action on the walls of the blood vessels, as is assumed by most writers, hence we were constrained to look further, and found that the coagulation

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time was reduced to from one-third to one-half or more after the hypodermic administration of pituitary extract.

We also made a study of the blood pressure before and after the administration of the drug. This was done in the hope that we might add something to the knowledge of the blood pressure of children in normal health, and also to study the effect of the drug on the blood pressure. The systolic and the diastolic pressures were taken before and after the administration by the auscultation method. A mercury sphygmomanometer of Stanton, with the modified cuff for children, as suggested by Gerstley, was used.

The following table of cases of tonsil and adenoid operations summarizes our results:

Serial Number	Michael Reese Hospital Number	Name	Sex	Age Years	Dose of Pituitary Extract Minims	Coagulation Time Before Administration Minutes	Coagulation Time After Administration Minutes	Systolic Blood Pressure Before Administration	Diastolic Blood Pressure Before Administration	Pulse Pressure Before Administration	Systolic Blood Pressure After Administration	Diastolic Blood Pressure After Administration	Pulse Pressure After Administration
1	70929	B. B.	F.	7½	12	3:15	2:00	90	40	50	95	40	55
2	70930	S. K.	F.	11	12	3:00	2:00	105	70	35	106	60	46
3	70944	F. R.	F.	4	12	3:00	2:30	80	50	20	84	45	39
4	71104	E. F.	F.	12	12	2:00	1:30	105	60	45	106	65	41
5	71121	H. B.	M.	12	12	2:00	1:00	125	85	40	115	70	45
6	71124	N. P.	M.	8	8	2:30	1:00	85	35	50	86
7	71258	S. S.	M.	2	8	2:40	1:45	110	75	35	117	75	42
8	71259	E. H.	M.	7	12	2:40	1:50	120	60	60	122	60	62
9	71261	A. B.	F.	6½	12	3:30	2:00	100	60	40	110	58	52
10	71384	R. B.	F.	16	16	2:00	1:30	120	75	45	125	75	50
11	71389	J. G.	M.	11	12	1:30	1:00	112	60	52	110	60	50
12	71390	L. M.	M.	8	12	1:40	1:00	103	50	53	103	50	53
13	71391	M. L.	F.	10	12	2:05	1:15	100	50	40	103	68	35
14	71406	I. B.	F.	11	12	2:30	1:30	95	50	45	105	55	50
15	71446	J. F.	M.	12	12	2:00	1:15	89	50	39	92	49	43
16	71447	B. B.	M.	6	12	1:10	1:15	68	45	23	70	50	20
17	71448	M. V.	F.	14	12	2:40	2:00	120	70	50	122	70	52
18	71460	L. S.	M.	21	16	2:30	2:00	130	65	65	130	60	60
19	71504	B. N.	F.	11	12	1:30	1:00	130	60	70	140	50	90
20	71505	G. G.	F.	5	12	3:00	2:40	80	30	50	85	50	35
21	71506	H. O.	M.	7	12	3:30	2:30	130	30	100	105	40	65
22	71573	L. M.	F.	5	12	3:00	1:40	105	60	45	95	70	25

23	71575	J.G.	M.	9	12	3:05	2:40	115	70	45	115	65	50
24	71728	N.R.	M.	11	12	3:20	2:50	110	55	55	116	55	61
25	71860	B.T.	F.	7	12	3:45	1:15
26	71837	B.S.	F.	5½	12	2:10	1:15	118	70	48	105	70	35
27	71908	J.H.	F.	8	12	2:40	1:20	110	70	40	105	55	50
28	71914	R.J.	M.	10	12	3:00	1:15	115	60	55	105	65	40
29	71977	M.C.	F.	21	16	2:15	1:09	125	85	40	110	65	45
30	72027	R.W.	F.	9	12	2:30	1:40	110	55	55	107	60	47
31	72033	K.L.	F.	6	12	4:00	3:00	105	50	55	100	50	50
32	72034	M.K.	M.	8	12	3:30	2:30	115	50	65	125	75	50
33	72046	B.E.	M.	11	12	3:15	1:15	105	45	60	120	25	95
34	72047	B.A.	M.	10	12	4:30	4:00	135	85	50	130	85	45
35	72048	F.S.	F.	12	12	2:30	2:20	115	65	50	120	70	50
36	72049	D.K.	F.	15	12	2:45	2:15	110	75	35	120	80	40
37	72051	M.G.	F.	9	12	2:35	2:15	110	50	60	100	50	50
38	72052	J.O'D.	M.	13	12	3:30	1:45	115	70	45	105	70	35
39	72093	G.K.	F.	9	12	4:30	3:30	105	45	60	100	50	50
40	72096	E.S.	F.	11	12	5:00	3:15	105	70	35	100	55	45
41	72098	L.R.	F.	7	12	4:00	3:00	130	82	48	130	60	70
42	72102	B.R.	F.	9	12	2:30	2:15	110	70	40	100	52	48
43	72107	I.K.	M.	11	12	3:10	1:30	110	75	35	130	70	60
44	72156	L.H.	F.	7	12	4:15	2:15	122	60	62	108	65	43
45	72157	F.H.	M.	6	12	5:00	1:00	105	75	30	104	65	39
46	72184	G.H.	M.	20	16	3:30	0:45	120	60	60	115	65	50
47	72223	T.S.	F.	5	12	3:45	2:10	99	55	44	102	60	42
48	R.K.	F.	11	16	4:00	1:00
49	B.B.	..	9	16	2:20	0:30
50	H.L.	..	29	16	2:00	1:02	130	110	20	136	110	25

Our cases, with the exception of three, were children aged from four to twelve years, to whom twelve minims of the drug were given hypodermatically. The influence on the coagulability of the blood was profound. It was reduced from one-third to one-half. In some cases a greater reduction was noted. For instance, one case required four minutes before the drug, and fifteen minutes after the administration, one minute. Another was reduced from three and one-half minutes to forty-five seconds, and still another from two minutes and twenty seconds to thirty seconds, and so forth.

The results of our observations of the blood pressures are noted below, under conclusions.

TECHNIC OF ADMINISTRATION.

Pituitary extract was administered hypodermatically in the dose of twelve minims to children and fifteen minims to adults, not less than fifteen minutes before the intended anesthetic. The coagulation time was taken before and again after the fifteen minute interval. The blood pressures were taken at the same intervals. The coagulation time was determined in the early cases with the Brodie and Russell coagulometer² and the remainder of the cases by the drop on the slide method.

CONCLUSIONS.

1. The coagulation time of the blood is materially reduced by the hypodermic administration of pituitary extract.
2. The hemorrhage following the nasal and throat operations is much reduced, especially those on the turbinal bodies.
3. The effect on the blood pressure of children is variable, as follows: Systolic pressure was increased in 55.31 per cent of the cases, reduced in 36 per cent, and unchanged in 8.5 per cent. Diastolic pressure was increased in 35.5 per cent of the cases; reduced in 35.5 per cent, and no change in 29 per cent. Pulse pressure is increased in 61 per cent, and decreased in 39 per cent of the cases.

REFERENCES.

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2. *Journal of Physiology*, 1897, Vol. xxi, p. 403.

XXV.

TUBERCULOSIS OF THE LARYNX.*

By J. HOLINGER, M. D.,

CHICAGO.

The pathology of laryngeal tuberculosis, the symptoms, the course, the prognosis and therapy depend mainly on the general condition of the patient, particularly on the condition of his lungs. It is, however, questionable whether we are justified in considering it, under all circumstances, as a secondary disease to tuberculosis of the lungs; in other words, there are few, if any, cases known where the lungs were free from tuberculosis and remained so. In 1902 and 1903 I observed such a patient, whose larynx suffered from microscopically diagnosed tuberculosis, yet he had no fever, no dullness or râles over his lungs, no bacilli in his sputum; in other words, there was at the time no affection of the lungs that could be diagnosed. Yet in 1905 he died of a very rapidly progressing tuberculosis of the lungs. His larynx remained cured to the end.

The only case of apparently primary tuberculosis of the larynx observed by the writer took such a course later on as to make it highly probable that an undiagnosed and undiagnosable focus of the disease existed at the time of observation.

In the large number of cases with fair general condition and slowly progressing or even stationary tubercular disease of the lungs, the symptoms of hoarseness and pain in swallowing are well known. Not so well known is the changeability of these symptoms, so that for days and weeks they seem to be entirely absent, and the patient and the doctor think the disease cured, until an examination reveals the great extent and destruction caused by the disease. This is a warning that

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the larynx of tubercular patients ought to be examined much oftener. Early discovered lesions give a better prognosis and better results from treatment. Furthermore, it is a fact that the elimination of a focus in the larynx has a good influence on the course of the lung trouble.

The diagnosis is usually not difficult. The subjective symptoms are pronounced. The objective examination by direct and indirect laryngoscopy reveals swellings or ulcerations, which may be found extending over the whole of the larynx, or, more frequently, over certain parts, the rear margin of the entrance of the larynx, or over the vocal cords, the epiglottis, or the space below the glottis. The sequence with which the localities of predilection are named is of importance. In the first place, the rear margin of the entrance of the larynx is given. Infiltrations of this part were often found when all the other parts of the larynx were free. Swellings of this part were also found combined with affections of other parts; for example, of the vocal cords. When, however, in such a case, the question of differential diagnosis from syphilis or carcinoma comes up, the infiltration of the posterior part of the margin of the entrance of the larynx speaks in favor of laryngeal tuberculosis. This point is so much more valuable, as it will be found to hold good, especially in early cases, when the differential diagnosis is often at the same time difficult and important. Of course, the other means of diagnosis, von Pirquet's test, Wassermann's test, microscopic examination, must not be neglected.

To illustrate some unusual features, the following history is given:

Mr. Z., fifty-eight years old, has lost several brothers and sisters from consumption. He is not very tall or strong. He has been hoarse since one year ago; absolutely aphonic for five months, and has complained during the last two weeks of difficulty in breathing. Otherwise, he feels perfectly well, and has not lost a day in years from his business. Examination of the larynx was easily made, and showed the cavity of the larynx concentrically narrowed to the size of a thin pencil by a hard swelling of the whole wall. The vocal cords could not be seen. The walls were irregular and of the same color as the pharynx—pale pink. The microscopic examination of a piece of the wall showed the presence of an inflam-

matory new formation. This finding was unsatisfactory. A larger excision with a Landgraf double curette was pronounced typical carcinoma by another pathologist. At the same time the condition of the patient improved under potassium iodid. Another specimen finally was pronounced tuberculosis, with scant but unmistakable tubercles. The wounds left by the different excisions healed promptly and completely. Therefore, the curette was used freely on both sides, and on the rear wall, until the normal width of the larynx was reached. The vocal cords were normal as to color and configuration. The left one was slightly paralyzed, but the voice at once became clearer. The patient made a quick recovery and was well for several years; then he developed tuberculosis of the lungs. The voice remained clear up to a week before death, according to information from his daughter.

The first point of interest to me was the finding of a large fibrous tubercular tumor of the larynx, whose walls were so thickened by a hard swelling as to have an opening of only the size of a small pencil, causing dyspnea. The patient's larynx remained well after the excision up to within a few days before his death.

The second point concerns the prognosis and the therapy. The prognosis of tubercular laryngitis depends entirely upon the condition of the lungs. It is good if the disease of the lungs either improves or at least does not progress; for example, a teacher whom I saw first in 1898—absolutely aphonic, with swellings of her arytenoid cartilages, and red, swollen, partly paralyzed vocal cords; has been teaching ever since—about three months after I first saw her. Other similar cases might be mentioned.

In the therapy, the first requirement is absolute rest of the larynx. The patient is not allowed to talk a word. Great improvement and even cures have been noted from this practice alone. Precautions ought to be taken that the psychic effect, the depression, does not become too great. In special sanatoria this can be done more easily and more effectively. Unfortunately, few of them are available to the middle and lower classes. Germany has found ways to overcome this difficulty. The results are correspondingly better. Local applications of lactic acid, thirty, fifty and seventy-five per cent, are often of benefit, if the patient is willing to submit

to the pain and discomfort they involve. At the same time the pharyngitis which is always present must not be overlooked.

Different forms of the galvanocautery, the simple stab cautery or the multiple cautery, also give very good results. In extensive applications of this agent the reaction is often quite considerable. A certain reluctance to use the knife and the curette was felt, dating undoubtedly from the time before the cocain and adrenalin. In 1913 Arnoldson¹ published a strong appeal in favor of the simple surgical methods of dealing with the different swellings. My experience in the above described case, and in others, spoke certainly in favor of this advice. The advantages are numerous. First, there is comparatively little hemorrhage, and the reaction, which is often quite serious after extensive cautery, is entirely absent. The young lady whom you saw tonight came to me absolutely aphonic. The epiglottis was at least one centimeter in thickness. I amputated the epiglottis two weeks ago—on a Saturday. On the Monday after that operation she went to work again. Her voice began to improve at once, though the larynx is not quite well.

Whether direct or indirect laryngoscopy is preferable is a matter of taste. Direct laryngoscopy gives better access to the diseased parts; indirect is easier on the patient, which advantage must not be underestimated in patients who are so easily affected by psychic influences.

An important point is finally the overcoming of the difficulty in swallowing, which often occurs in the later stages of the disease. The question is of great importance, as many patients do not eat because swallowing hurts them so much that they avoid it at all costs. The consequence is that they lose weight and ground very fast. The simplest means by which to overcome that is by the use of the narcotics and local anesthetics—morphin, cocain, orthoform, anesthesin, and nerve blocking. Morphin, cocain, orthoform and anesthesin have certainly all some influence on the heart, which is absent in nerve blocking. Nerve blocking consists in the injection of alcohol in the vicinity of the superior laryngeal nerve. Since the procedure is very simple, it must be recommended in preference to the narcotics and local anesthetics. But under all circumstances we must not forget that all this will not

have any curative effect. If we do not at the same time apply any local surgical measures, we simply keep busy, doing nothing for our patient.

External surgical measures, tracheotomy and laryngofissure, have both been used by the writer in a few cases. They invariably leave the patient in a much worse condition than he was before, either generally or locally, often both. Even laryngectomy was advised by some authors. Whether the life of these patients is prolonged thereby, I do not know; surely their sufferings are. Whether radium, X-ray and sun rays will accomplish all that is claimed is questionable; we are inclined to expect more of the things we do not know anything about. The well known surgical procedures have the advantage that we know what we are doing. It is true, they require experience in the diagnosis, and skill in their performance, but those are qualities that we can and have to acquire.

REFERENCE.

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XXVI.

CHOANAL FIBROMA.*

BY NORVAL H. PIERCE, M. D.,

CHICAGO.

These growths constitute a group which, pathologically and clinically, are rare but interesting. There is some discussion as to their place of origin. Panse, in 1873, was the first to draw attention to these growths; and other accurate observers, among them Zarniko, believe that they originate somewhere around the rim of the choanæ, while Killian believes that they have their origin within the antrum, and gain exit to the postnasal space through an accessory opening. Kubo, a scholar of Killian's, made a broad dissection of the antra in several of these cases, and proved that at least some of them do spring from the antrum and escape through an accessory opening in the postnasal space. It is probable that they may originate both around the rim of the choanæ from the septum, and the posterior end of the middle turbinated body, as well as from the accessory sinuses. They differ altogether in structure from the myxomas or common polypus, inasmuch as they are made up of true connective tissue covered by several layers of epithelium.

Zarniko makes the point that in one of his cases well defined bone formation was present, which would at once take it out of the class of mucous polypi. They have always one peculiarity in common, namely, the very fine pedicle by which they are attached to their base.

They may grow to a very large size. Stork reported a case that measured nine centimeters long and was ten and one-half centimeters in circumference.

Zaufal reported a case which measured eleven centimeters long, six centimeters broad, and three and one-half centimeters

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thick. The circumference was fourteen centimeters. One of my cases measured six centimeters long from the point of insertion to the end of the growth, and at its largest portion measured three centimeters.

They belong to the type known as soft fibromas.

DIAGNOSIS.

The diagnosis has to be made between typical postnasal fibromas, pseudopostnasal polypi, and sarcoma of the postnasal space. As is well known, the true typical postnasal fibromas occur most frequently in males, between the ages of eighteen and twenty-five. It is a remarkable incidence that the three cases that I have seen in the last three years occurred in females, one in a Japanese girl of twelve, the other two in young adult females.

The true typical postnasal fibromas at their commencement have a tendency to bleed excessively, whereas the hemorrhage from the choanal fibromas is negligible.

It has been said that another point in differential diagnosis is that the true postnasal fibromas have a tendency to form adhesions between the surrounding tissues, while the choanal fibromas do not. In one of my cases I could positively prove the adhesion which had taken place between the body of the tumor and the posterior wall of the postnasal space.

The point of origin is another very valuable asset in differential diagnosis. It is at times difficult, when the tumor has reached a size that completely fills up the choanæ and projects down into the postnasal space, to ascertain this by posterior palpation. We can, however, by means of the use of Killian's long intranasal speculum in many cases discover the point of origin, either from an accessory opening or from the rim of the choanæ.

TREATMENT.

On account of the delicate pedicle by which these tumors are attached at their base, their removal is very easy. Lange's hook is of especial use in the removal of these tumors. By means of this hook, which is inserted into the anterior naris and made to engage the pedicle, the tumor can be severed from its base very easily. It is then either caught in forceps and dragged through the nose and delivered anteriorly, or allowed

to drop into the postnasal space. The snare may also be used, inserted through the anterior naris, while a finger in the postnasal space directs the snare over the body of the growth. If it is not too large, traction is made and the tumor is delivered through the anterior naris. I have also removed one of these tumors by means of a large-sized adenoid forceps, the tumor being delivered through the postnasal space.

The prognosis as to recurrence is very good. In the three cases I have had, one has not recurred after three years, one after eight months, and the other after three months.

The remarkable fact is that these tumors are so frequently mistaken for true postnasal fibromas by specialists of good repute. The differential diagnosis is easy, if we call to our aid the few points that I have already named. The mistake that is made is depending on palpation for diagnosis. These tumors may be quite fixed in position and appear quite hard to the touch—quite as hard as a true postnasal fibroma at its beginning; but with our probe we can ascertain that they do not spring from the basilar cartilage, and by means of the intranasal speculum we can most frequently discover their point of origin in the choana.

XXVII. -

HAMMOND'S MASTOID SKIN GRAFTING.*

(A PRELIMINARY REPORT.)

By FRANK ALLPORT, M. D., AND ALEX. S. ROCHESTER, M. D.,

CHICAGO.

Among the chief objections to the radical mastoid operations are:

First, the tedious healing and painful dressings.

Second, the not infrequent incomplete bone covering, constituting uncured cases.

Third, the thin and poorly nourished epithelium often present after cases are really cured, frequently occasioning the deposit of scabs, pus, etc., in the concavities of the large bone opening, necessitating indefinite treatment and care.

Fourth, the nonclosure of the tympanic opening of the eustachian tube, rendering possible more or less dripping from the meatus, thus producing unsatisfactory results; and

Fifth, depreciated hearing, owing to the formation of thick cicatricial tissue over the oval window.

These are some of the charges brought against this operation, and these are the things that can be usually avoided, if proper skin grafting is employed, according to the advocates of the procedure. We cannot deny that any or all of these misfortunes may occur; but whether they can be avoided by skin grafting or not, is another question. We have nothing to say against skin grafting; but if it will accomplish these happy results, why is this aid to healing not more generally adopted? Of course, this question is not at all conclusive, for, as we all know, many good things knock for years at the door of success before being admitted; but still, after being before the observation of mastoid surgeons for years, and occupying

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still an insecure position, in their minds, its great utility is certainly more or less clouded in doubt. The fact is, that whatever may be the merits of skin grafting after radical mastoid operations, the assertion can be safely made, that it is used by only a small percentage of operators. Many of those who condemn grafting have never used it, and are contented to abide by the decision of skillful operators who oppose the procedure, or who at least have good results without it, and who, therefore, see no reason to lengthen their operations by a tedious, puttering, and sometimes unsatisfactory process. Many of those who do not graft have used it on a few cases, been disappointed, and have abandoned it, forgetting the fact that this kind of work requires much practice before its real value becomes apparent. A few failures must be experienced before success will be attained. Fortunately, failures in grafting do not mean real operative failures; because, if the graft does not live, the operator can go right on treating the case just as if the grafts had never been placed. It is, therefore, perfectly safe to at least give skin grafting a thorough trial, and to only abandon its use after an operator becomes convinced from personal experience that it is, at least for him, a useless procedure.

The chaotic condition of this subject is perhaps responsible for the insecurity of its position, as apparently no two men operate the same way. Some men graft immediately after the radical mastoid operation is performed, while others wait for from six to ten days, and then open the wound and graft. This operative distinction is not a mere notion or fancy—it is a principle; as those who do secondary grafting, do so because they do not believe that grafts can be at all depended upon to grow upon newly cut bone. Nevertheless, those who advocate primary grafting go serenely on their way, grafting primarily, and claiming thoroughly satisfactory results. Some operators believe that grafts grow best on thoroughly smoothed bone, where the graft and the bone surface come in perfect apposition, while others feel that smoothed bone has temporarily lost its highest vitality and does not furnish the best soil for transplanted skin. This has been utilized as one of the objections to using electrically driven burs in mastoid operations, as it is claimed that the bur revolves with such rapidity that superficial bone necrosis is produced, which con-

siderably retards healing and renders grafting unfeasible. We have used the bur many times in mastoid work, and have a few times seen this occur. The first dressing reveals a superficial black necrosis, and an exceedingly offensive odor. Under such circumstances strong antiseptic daily dressings should be made, and in a short time all evidences of this temporary embarrassment will subside. We think there is no danger of this complication, however, if the operative area is kept moist by droppings of sterile salt solution. It is only where the bone becomes dry and overheated that necrosis is produced. Therefore, if the surgeon proposes to graft primarily, and a rapidly driven bur is used, the bone should be kept constantly moist, or the grafting will be a failure. We are confident that smoothed bone is better adapted to grafting than roughened bone, and no instrument leaves such a beautifully smoothed cavity as the electrically driven bur, but great care should be taken to keep the bone moist. We think that a properly excavated and prepared bone cavity has much to do with the successful termination of cases that have been skin grafted. All necrosis must be removed, the eustachian tube must be thoroughly curetted and scraped, the inner tympanic wall must be carefully but thoroughly curetted, the upper meatal wall must be removed so that it is on a level with the tegmen, and the posterior meatal wall or ridge must be cut away so that the ridge is entirely removed between the meatal floor and the mastoid floor, from the external surface of the bony opening to the point of danger for the facial nerve.

Some operators use large grafts and others use small ones. Large grafts, however, are practically used by all surgeons when the skin is inserted into a retroauricular wound, small grafts being reserved for those cases where the transplantation is performed through the meatus, when time has shown that complete dermatization of the cavity should not be expected. Some operators systematically graft through the meatus, about ten days after the mastoid operation. Under these circumstances, of course, only small grafts can be used; and they are usually rather difficult to place and to hold in position, owing to the contracted space, varying illumination, and the tendency to curl at the edges. The ease of placing these small grafts through the meatus can be increased by resorting to the following procedure: After gently cleansing

and drying the cavity through the meatus, and waiting until all bleeding and oozing has ceased, the little graft is placed on a glass slide, skin side up. A very slender hollow canula or glass tube, bent or straight, with an air bag attached, is made to gently suck up the graft upon its end, by cautiously using the air bag. Under the best possible illumination, the canula loaded with the graft is passed through the meatus (which has, of course, been enlarged by the previously performed radical mastoid operation) to the granulating bone surface. The bag is now compressed gently, and the graft is blown into position, after which the canula is withdrawn, the graft is gently adjusted with a delicate probe, and other grafts placed in position in the same manner. If the grafts persistently curl at the edges, it is better to leave them alone and to continue placing more grafts, even if they overlap here and there. When the granulating surfaces are covered, the cavity should be gently packed with quite small cotton pellets, dusted with something like aristol. Some greased gauze should be gently placed over the pellets, and brought out through the meatus. The first dressing should occur in about four days, and the packings should be loosened by peroxid of hydrogen and warm sterile water. No force should be used. Gentle daily dressings may now follow, with cleansings, boracic acid powder, etc., until the case is finished.

It will thus be seen that there is a wide diversity of opinion concerning the methods of grafting after mastoid operations; and further differences of views become still further apparent, when we come to consider the varying steps of the operations. We do not intend delving deeply into the different methods of grafting. There is much that may remain unsaid, as the fundamental steps of such operations are too well understood to warrant further repetitions. There are, however, certain individual ideas, entertained by experienced operators, to which we will endeavor to briefly refer.

There can be no question that grafting large grafts immediately after the radical mastoid operation is the ideal procedure, if grafting is done at all; the only question to be settled is, whether such grafts usually live, and whether the process of healing is assured and quickened thereby. The operation is not difficult (after a little experience), and it is

certainly better for all concerned than any kind of secondary grafting, provided it can be shown to be successful.

There are said to be two principal objections to primary grafting.

First, skin does not adhere well to bare bone, and little patches of granulations should be present, if life to the graft is to be confidently expected.

Second, when all necrosed bone is not removed at the mastoid operation, such centers of disease may create considerable disturbance to bone and meninges, if immediately covered over by grafted tissue.

The latter of these objections seems to us quite insignificant and unreal, as a thin graft of skin is hardly strong or stable enough to retain the products of bone necrosis. The former of these objections, however, seems to be more noteworthy in its character, and yet we have the evidence of such men as Dench, Hammond, Welty and others, to the effect that they are increasingly satisfied with the success of primary skin grafting. We are of the opinion, therefore, that mastoid operators should, by personal experience, convince themselves as to the utility of grafting, and should be quite sure of the ineffectiveness of primary grafting before dooming it to abandonment. If primary grafts do usually live, and if they shorten the healing process, they certainly possess the advantageous quality of rendering unnecessary secondary surgical procedures. We, therefore, urge that they be tried first, and be abandoned only for good reasons, especially as it is just as easy to insert primary as secondary grafts—indeed, the process is practically identically the same.

The points to be emphasized in ordinary primary skin grafting are as follows:

First, remove all necrosis and make the cavity as smooth as possible.

Second, remove all possible soft tissue from the inner wall of the tympanic cavity.

Third, thoroughly curette the eustachian tube as far as the isthmus, and rim out its tympanic orifice.

Fourth, stop all bleeding and oozing by forceps, pressure, firm packing, peroxid of hydrogen, hot water, hot one per cent solution of the chlorid of calcium, adrenalin, patient waiting, etc. Neglect of this direction will spell failure.

Fifth, large thin grafts should be cut and floated from the razor, cut side down, to a glass slide, by warm salt solution and a needle, keeping the edges uncurled, if possible. Some operators float the grafts in a basin of warm salt solution, cut side down, and pick them up on a spatula or glass slide just before using.

The body area from which the graft is to be cut should, of course, previously have been thoroughly cleaned with normal saline solution and soap, and bandaged.

Sixth, the graft should be slid from the glass slide by means of normal salt solution moisture and a needle or other suitable instrument, being careful to frequently smooth out the curled-in edges of the graft. It is better to line the anterior and interior portions of the cavity first, and then add more grafts as seen necessary.

A portion of the graft should be firmly tucked into the eustachian tube. The slight overlapping of one graft by another is harmless; the overlapped portion will simply slough off. The curling in of the edges of the graft cannot always be controlled, and a graft need not be condemned on this account.

Milligan proposes a rather novel method of placing the grafts. He fills the cavity with a warm salt solution and floats a large graft upon it, cut surface down. He then sucks out the salt solution by means of a pipette, and steers the graft into position with a needle or some similar instrument. He claims there is less tendency for the edges to curl, and that it is easier to place the graft.

Whatever means is adopted to place the grafts, care must be taken that they lie as smoothly as possible, and that they come into direct contact with the bone. The smooth pointed needle, or a smooth rounded probe, or instrument, such as suggested by Ballance, are best adapted for this purpose. The instrument should pack down, but not displace the graft. Blood, or other moisture, or air, will sometimes accumulate under the graft, and may be sucked out by the careful use of a curve pointed pipette.

It seems better not to graft the posterior and lower portions of the cavity, as it produces too large a cavity. Besides this, these portions of the excavation heal rapidly through the agency of well made meatal flaps. The important thing is to

cover as much as possible, the tegmen, attic, tympanum and antrum, so that these diseased centers which are remote from the meatal flap may be speedily covered with new and healthy tissue. We materially assist in the dermatization of the upper portion of the cavity by the nature of our flap. It is a modified Panse flap. Panse split the meatus in the middle; we split it as far down as possible, thus giving all the flap to the upper wall; the lower does not need a flap, it takes care of itself, and always skins over. Our flap is an L flap; Panse's flap is a T flap. That is all the difference. The Allport divulsor enables one to make the split accurately, wherever it is desired.

Philip Hammond, of Boston, has lately been using a method of applying a graft to the mastoid cavity that commended itself to our reason as being simple and efficient. We will use his own words to describe the procedure: "A large thin graft, two inches or more square, is cut from the thigh with an ordinary amputating knife. The skin is immediately spread out on the place from which it was cut, exactly as if it had not been severed from its attachments. A square of crepe de lise is then laid on it, and the skin and crepe together removed from the thigh. This simple device absolutely prevents rolling or curling of the edges. A piece of ordinary gauze—or, rather, several layers which have previously been rolled into a cone, are then placed under one corner of the crepe and skin, in such a manner that the entire apex of the cone and at least one side is completely covered with skin, the raw side out. One side of the cone is purposely left free of skin, as this side goes into apposition with the anterior wall of the canal, which is already covered with epidermis. The cone, or plug, is then introduced through the meatus, the point guided toward the middle ear, and then the gauze of the plug gradually fed into the cavity, expanding and completely filling it.

"It must be remembered that the graft is adherent to a thin, smooth, silky crepe, and the pressure of the mass of gauze causes this to reach every part of the cavity, even where the meatus is much smaller than the inside, the gauze expanding in much the way that a bristle probang does. This gauze is allowed to remain undisturbed three or four days, at the end of which time it is gently removed. The crepe de lise

comes with it, but the skin never. In the interval it has become firmly attached to all denuded surface. There are naturally some loose ends where the graft is superimposed upon the epidermis of the meatus, and there are necessarily reduplications or folds in the graft, as it is impossible to adapt a flat surface to the inside of a sphere without such. These loose pieces finally slough out, and during the process there is a very active suppuration to deal with. In most of the cases the superficial layers of graft are cast off, but underneath there is the firm pink appearance of new skin, rapidly paling out. Immediately following the removal of the gauze-plug it is possible to demonstrate that the graft has 'taken' by pulling upon it. It stands a very considerable strain, and if we succeed in detaching a piece, it causes bleeding, showing that it is definitely attached to its new resting place."

We would say, that "crepe de lise" is a thin, silky goods that can be purchased at any good dry goods store.

Concerning the steps to be taken after the graft (or grafts) has been placed in position, it is, of course, evident that if Hammond's method is used, no other packing is required, as the gauze which is introduced with the graft, and its crepe de lise backing, will be quite sufficient.

If, however, this method is not used, most operators resort to small cotton pellets, thoroughly dusted with something like aristol or xeroform. Thorough dryness favors the life of the graft. The number of pellets used should be noted, as this is of assistance in feeling certain that all have later been removed. The first pellet should be placed in the anterior inferior angle of the cavity, in the vicinity of the eustachian tube, and each pellet should be carefully placed so as not to disturb the graft, which can, of course, be most easily displaced. When the cavity is filled, a strip of greased gauze should be placed over the pellets and its end brought through the meatus and the wound sutured.

Some operators have peculiar methods of their own in preserving the grafts. Politzer, for instance, covers the grafted area entirely with tin foil; while Ballance uses very fine gold foil instead; Reinhard uses fine silk tissue as a covering. Some use pure cotton for pellets, while others cover the little pellets with gauze. Denker uses no pellets at all, but merely drops

a little loose cotton into the cavity, which is removed at the first dressing; others use loose gauze.

Unless otherwise indicated, it is recommended that the pellets should remain undisturbed for from five to seven days, at which time the packing should be gently soaked with peroxid of hydrogen, and all loose pellets removed. No force should be exerted. The cavity should now be gently syringed with a mild, warm, antiseptic solution, and then gently and loosely packed with greased or dry gauze. There is usually much odor present at the first dressing, and perhaps at the first two or three dressings, owing to the death of unadhered skin; but this soon passes away. After the odor subsides and the pellets, and gold, tin or silk coverings have been gradually and carefully removed, as little syringing should be done as possible, the cavity daily treated after the dry method, and kept powdered with boracic acid powder packings. As soon as possible all packings should be suspended, as any pressure is detrimental to the life of the graft. In cleansing the cavity at the first dressing, all of the grafts may be alive, or some of them may be dead; but at all events, even if a graft is dead when it is removed, a little island of new epithelium will usually be found underneath it, from which dermatization will proceed. In such cases of partial failure, subsequent grafting of small patches can be easily accomplished through the meatus. At all events, and under almost any circumstances, the process of healing will be probably hastened by any kind of a proper grafting process.

To Ballance must be given the credit of bringing prominently into notice systematic secondary grafting. He performs the original operation, and closes the wound with sutures. In ten days he opens the wound (after several thorough cleansings), and takes great care not to injure the newformed granulations, or the newly attached meatal flap. The bleeding and oozing are stopped by hot water, adrenalin, etc., and then the large thin graft (or grafts) is placed, as has already been described. Ballance's use of gold foil, cotton pellets, etc., has also been mentioned. He then sutures, etc., and does not disturb the wound for one week, when it is gently dressed, removing such packings, etc., as come away easily. Subsequent dressings are made every two or three days. We have seen Ballance's results, and it is most satisfactory to see how

beautifully and almost invariably the grafts live. But there is, of course, the great objection to the secondary anesthesia and operation.

Some operators believe that no graft should be placed over the inner tympanic wall, because of the possibility of deafness resulting from covering the oval and round windows; others believe in covering the wall, but leaving an aperture through the graft to accommodate the oval window. The general consensus of opinion, however, is that, if great care is taken to place a graft over the inner tympanic wall that is exceedingly thin, its presence will not impair hearing, but may even improve it. Milligan has collected one hundred cases of grafting after radical mastoid operations, and has tested them all carefully, both before and after operation, and has kept most of them under observation for months and years. His results showed sixty-four per cent materially improved in hearing, twenty-four per cent unimproved, and twelve per cent were made worse. Milligan is not alone in his conclusions; they are shared by many experienced surgeons.

There is no diversity of opinion as to whether exposed brain or sinus dura should be covered by a graft or not. Many surgeons advocate covering these soft tissues with a graft, provided they are reasonably certain that all diseased conditions have been removed. Most surgeons think, however, that while there is no objection to grafting on bone, that when the brain or sinus are exposed, it is better not to attempt to cover them with grafts but to leave these areas uncovered.

Certain surgeons have devised peculiar methods of securing grafts, that are interesting if not useful. For instance, Waggett varnishes the grafting surface with "Newskin," which, he claims, prevents curling of the edges and favors an easy manipulation of the graft. Wingrave uses, in the same way, a ten per cent solution of acetone in collodion. Deanesly uses sticking plaster on the skin before making the graft. Any or all of these methods may not only be interesting but useful, but we have had no experience with any of them, and so cannot express an opinion.

Any radical mastoid operation, with or without grafting, will be followed, more or less, for some time by an accumulation of debris, consisting mostly of desquamated epithelium, especially in the most concaved and remote portions of the

bony crater. These accumulations do not mean that the case is not cured; it means that the new skin is thin and poorly nourished, and throws off scales, etc., which, if not cared for from time to time, will provoke a pus deposit and present a most undesirable appearance. These cases should be thoroughly cleansed from time to time by syringing, etc., and a two per cent solution of salicylic acid in alcohol freely poured in the ear. Such conditions will gradually pass away, as the health of the new skin becomes established, but while it lasts the ear should be cared for, as just described.

About six months ago we resolved to give the method of grafting used by Philip Hammond, of Boston, a fair trial in selected cases. This method appealed to us as being an efficient and comparatively simple one. We have used it in six cases, and herewith beg to submit our results as far as we have gone:

Case 1.—Girl, aged six years. Right ear. Foul discharge for two years. Adenoids removed. Ear treatment used for two months. No result. Radical mastoid operation performed, with Hammond's skin graft. First dressing in five days. Foul odor and much discharge. Part of the graft adhered, and part came away. Subsequent dressings were done every other day for two weeks, and then once a day. Healing occurred in about the usual time, namely, eight weeks.

Case 2.—Girl, aged seven years. Left ear. Discharge for four years. Very foul odor. Two months' treatment of no avail. Radical operation performed. Hammond's graft used. First dressing on fifth day. Slight discharge and odor. Entire graft preserved. Subsequent dressings every other day for two weeks, and then every day. Case cured in one month.

Case 3.—Boy, aged five years. Right ear. Foul discharge. Adenoids removed. Mastoid pain and tenderness. Radical operation. Hammond graft. First dressing on fifth day. Much discharge and odor. Entire graft came away. Healing in about ten weeks.

Case 4.—Girl, aged five years. Right ear. Discharge very foul for one year. Some surgeon had performed an acute mastoid operation about one year ago, but discharge continued. Adenoids removed. Treatment for two months availed nothing. Radical operation. Hammond graft. First dressing in five days. Some graft remained. Healing in five weeks.

Case 5.—Boy, aged six years. Right ear. Foul discharge for two years. Two months' treatment. No result. Adenoids removed. Radical operation. Hammond graft. First dressing on third day. Slight odor and discharge. Graft entirely saved. Left hospital on tenth day. This boy came from filthy and shiftless surroundings. The importance of postoperative care could not be taught. Did not see boy for seven days after leaving hospital, and after this constant care was impossible. At one dressing, two flies, one flea, and a bedbug were found in the ear. We are confident if this case could be properly cared for, a cure could be effected, but as it is, the case seems hopeless; but the graft was a success.

Case 6.—Girl, aged ten years. Left ear. Foul discharge for seven years. Cholesteatoma. Adenoids removed. Radical operation. Hammond graft. Graft was thick, but it adhered well. First dressing in three days. Cure in four weeks.

It seems difficult to secure ideal grafts of proper size and thinness. This, however, can be overcome by a proper razor and experience. Some surgeons feel that grafts will not stand much handling, but this has not been our experience. While, of course, we advocate care in handling grafts, it may be said that the graft, in our series of six cases, which did the best, was the one which was subjected to the most manipulation. This was in case 6, where, owing to the bad conduct of the patient under anesthesia, considerable haste was observed in operating. In consequence, a thick graft was cut, with considerable subcutaneous tissue attached. This tissue was cut off by scissors and scraped off by a scalpel. Manifestly, considerable violence was employed, and yet the graft lived completely, and the case was cured in four weeks.

We believe that the first dressing should be done in two or three days after the operation, instead of waiting five or six days, as most surgeons advocate. If done early, and with extreme care and delicacy, and without using any force whatever in withdrawing the packing, the cavity does not become so wet and foul as it does if five or six days are allowed to elapse. We believe also, that after the first dressing, subsequent dressings should be made every day. Of course, six cases are quite insufficient evidence upon which to base a dependable opinion, and yet they have distinctly encouraged us to continue, for a time at least, the use of the Hammond

method of grafting, and we certainly are convinced that any doubts as to the livability of grafts on newly cut bone should be abandoned. All grafts but one lived more or less, and the time necessary for a cure certainly was shorter than our average cases, where no grafts were used.

SHORT TABULATED REPORT.

Case.	Did graft live?	Time of healing.
1.	Partly.	Eight weeks.
2.	Yes. Entirely.	Four weeks.
3.	Not at all.	Ten weeks.
4.	Partly.	Five weeks.
5.	Yes. Entirely.	Case uncured, owing to lack of parental coöperation.
6.	Yes. Entirely.	Four weeks.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOTOLOGY.

Meeting of December 3, 1914.

**Metastatic Conditions Complicating Suppurative Otitis Media, With
Especial Reference to Secondary Inflammatory Processes
in the Joints.**

DR. SEYMOUR OPPENHEIMER calls attention to the general symptomatology common to all cases of infective thrombosis, and in addition cites those characteristics of metastases located in the chest, the abdomen, the cranial cavity or the joints.

Cases are observed with so slight a clinical picture from the ear, that because of the temperature curve, they are wrongly diagnosed as typhoid fever. The author saw two cases, thus admitted to the medical service, in which, however, positive blood cultures eventually led to a correct appreciation of the lesions, and sinus thromboses were discovered and operated upon.

The author is inclined to regard endocranial complicating lesions, not in the light of true metastasis, but rather as lesions produced by contiguity. Regarding endocarditis, he points out the difficulty of recognizing this condition before death, and cites two cases in point which terminated fatally. He refers to the cases of hepatic abscess reported by McCuen Smith, who, however, did not demonstrate the presence of thromboses in his cases, nor give the report on blood examinations. Oppenheimer is at a loss to explain these cases as metastatic lesions, unless sinus thrombosis and a general bacteremia is conceded. So, also, cases of appendicular inflammation are to be explained. He saw three cases of acute gall bladder inflammation which supervened within two weeks after acute mastoiditis. No thrombosis was found, however, and no blood cultures were taken.

Arriving at the question of particular moment in his paper, Oppenheimer draws attention to cases of joint inflammation which resemble acute rheumatism, and which, not infrequently, occur in infective thrombosis. One or several joints, either with or without pus formation, is presented. Usually the characteristic symptoms pointing to an otitic basis are found, although it is noteworthy that the ear symptoms are of a very mild degree.

Acute articular rheumatism is conceded to be a disease, *sui generis*, of an infectious nature, whose causation is still a mooted question. When joints are involved, a number are affected, and the disease travels from one to another, seldom tending toward true pus formation. The peri- and endocardium are often involved. Rheumatism shows a tendency toward hereditary characteristics, toward relapses, and it tends to react to specific chemical agents. However, different views are held regarding rheumatism, and some of these Oppenheimer cites, conceding the common observation, that the most varied types of microorganisms are able to produce the typical picture of acute articular rheumatism. Finally, the author refers to the animal experiments, the results of which undeniably demonstrated that weakened pyogenic cocci can and do produce a picture of articular rheumatism.

Oppenheimer contends that if it is conceded that a disease appearing in the guise of an acute articular rheumatism can be caused by pyogenic cocci, then this can only take place provided such microorganisms find entrance into the blood stream in a weakened form; otherwise they would produce an entirely different clinical picture, namely, pyemia with abscess formation.

From all of the above, Oppenheimer concludes that we can comprehend how symptoms of joint inflammation may present as a complicating lesion and an overshadowing picture to a middle ear suppuration—the symptoms from the joint being so prominent as to completely obscure the original causative disease. He cites the following cases in point:

Case 1.—F. M., aged thirty-four years. A right-sided otitis media, which after myringotomy presented a scanty discharge for ten days. After one month suffered chills and rises of temperature and swelling of left knee. Ten days later the knee was pronounced to be a secondary arthritis.

Patient now is septic. There is no aural discharge, no mastoid tenderness, and no intratympanic evidence of mastoiditis. Some submaxillary and cervical lymph node enlargement. Left knee joint presents an acute synovitis and peri-arthritis. Blood culture positive in twenty-four hours, with forty-five colonies of streptococci.

The affected knee joint aspirated, evacuating a large amount of yellowish purulent material containing streptococci. Mastoid exenterated, sinus explored, and thromboses demonstrated. Jugular ligated and exsected. Repeated cultures from blood gave progressively lessening numbers of colonies to the cubic centimeter.

Ten days after sinus operation, temperature again rose to 103° , and pain and swelling appeared in the left shoulder. Patient died fifteen days after sinus operation.

Noteworthy among the portmortem findings was the evidence of vegetations on the mitral heart valve, smears from which gave streptococcus.

Case 2.—Female, forty-nine years of age. Chronic aural discharge for fifteen years, which ceased three months prior to coming under observation, at the time when a postauricular swelling presented. This swelling was reduced by local applications. One month later the ankle joint began to swell and, upon examination by patient's physician, a peri- and endocarditis with pleural exudate was found. Three weeks later the swelling of the ankle subsided, but recurrence took place after patient went to work. The patient then had dyspnea in addition.

Suddenly there presented itself pain in the ear and a chill and a rise of temperature to 104° . The next day a second chill and rise of temperature.

The local examination gave membrana tympani absent, some granulations in the middle ear, and slight odorous discharge. Mastoid not sensitive. No choked disc.

The operative findings revealed an extensive destruction of the inner table, particularly of the posterior fossa wall, with a large perisinus abscess. Sinus thrombosis demonstrable. Jugular exsected.

The patient progressed favorably for two weeks, and then symptoms of cerebellar abscess manifested themselves and a

large occipital lobe abscess was evacuated. Death followed. No autopsy.

Case 3.—Female, aged fourteen years. Admitted to hospital with diagnosis of acute articular rheumatism, which followed shortly after an acute suppurative otitis media. There was a history of an acute aural suppuration, lasting four weeks, which had occurred two months previously. For one month, chills almost daily, high temperature, sweating, and headache. Swelling of phalanges of left hand and of left hip joint and stiffness of neck. Blood culture positive. Examination shows absence of aural discharge, no mastoid tenderness. Beginning optic neuritis. Operative findings demonstrated a purulent thrombus in the sigmoid sinus and upper part of the jugular vein.

Case 4.—A. L., aged seventeen years. Came under observation with a very slight postinfluenzal, acute suppurative otitis media, the bacterial flora of which proved to be the streptococcus mucosus. Ten days later ear symptoms ceased, but a few days thereafter pain and swelling in the phalanges and wrist of the left hand developed, to be followed within a week by rapid development of involvement in both knees, both elbows, right hip and right shoulder joints. Aspiration of knee and shoulder joints, but no bacteria recovered. A severe bronchitis also developed. This condition continued for six weeks, during which time no ear symptoms presented; then mastoid tenderness, a sharp chill and temperature of 103° . The membrana tympani was intact, thickened and reddened. Tenderness and edema over mastoid. The operative findings give evidence of a beginning perforation of mastoid cortex and destruction of internal plate. Pus from mastoid gave streptococcus mucosus. Septic thrombus extending from sinus into bulb and into jugular to level of the thyroid vein. Jugular exsected. The patient recovered.

Case 5.—Male, four years old. Under treatment of his physician with swollen ankle, knee and both shoulder joints. Temperature varying from 99° to $102^{\circ} +$. Complains of pain in ear and shows a slight swelling of auditory canal. The mastoid slightly sensitive, and a scant amount of foul smelling discharge present. Ossicles absent. The patient has a chronic aural suppuration. The temperature is now 105° , preceded by chilly sensations. Operative findings demonstrate

a sclerotic mastoid, no free pus; sinus plate necrotic, and sinus covered by thick exudate and granulations. The sinus contained a firm, white, apparently well organized thrombus, streptococci being demonstrated in thrombus and vein wall. Jugular excised. Patient recovered.

These case histories demonstrate that an otitic sinus thrombosis may be masked by the symptoms of an acute arthritis. Oppenheimer believes that the toxins from the original infection are to be considered as the causal agents of the bacteria-free joint exudates, and he holds it superfluous to raise and discuss the question as to whether the joint involvements were primary, or whether they were truly sequelæ of the ear lesions.

The author concludes that if after an acute otitis, or during the course of a chronic suppurative otitis, symptoms of acute arthritis supervene, these must not be considered as primary, but should be looked upon as secondary metastatic complications resulting from an infective otitic phlebitis and associated general bacteremia.

He does not preclude the possibility of secondary joint lesions which follow a recognized ear lesion and sinus thrombosis, but draws attention to the fact that here another class of cases, different from those under discussion, is being dealt with. General practitioners of medicine should realize that an ear disease with a clinical picture and a symptomatology of so slight a nature as to be almost overlooked, may produce joint symptoms with a stormy clinical picture, and meanwhile the original lesion progresses and endangers the patient's life. In all joint lesions with a history of aural trouble, the possibility of these facts should be kept in view, so that a sinus thrombosis may not be overlooked.

DISCUSSION.

DR. JOHN ROGERS remarked that in his experience multiple suppurative processes of inflammation or pyemia following ear infections were quite rare. When this calamity did occur, it was well recognized by surgeons that the primary source of the disturbance must always be sought and relieved. In the presence of suppuration in the middle ear, this means that a radical mastoid operation must be performed at the same time that the abscesses in the joints or elsewhere are opened and drained. These pyemic conditions of severe grade are

much less difficult to diagnose than those of milder form which now pass under the designation of polyarticular rheumatism. There can be little doubt at present that a great many nonsuppurative and multiple inflammations of joints have a bacterial origin. They seem to be due to what is virtually a pyemic process, or an inflammation from some mildly pathogenic organism which originates in one of the many possible primary foci in the head, or genitourinary, or alimentary tract, and thence travels by the blood, with or without the intermediation of the lymphatics, to the joints.

The diagnosis in these cases is by no means easy, as in addition to the subacute or comparatively mild articular infections, there are many others which can only be explained as noninfectious or metabolic. To increase the difficulties of diagnosis, some of the chronic lesions of joints or cases of chronic rheumatism are strongly suspected of having a bacterial metastatic origin. Arthritis deformans, and even the Charcot joint, may belong in this class. As yet there is no method of accurately differentiating the pyemic from the metabolic arthritis; hence, in treatment, the best we can do is to search the body carefully for all possible sources of infection, and the ear is too often overlooked. Dr. Oppenheimer had pointed out that infection may exist in this region and give so few symptoms that it may readily be forgotten. If, however, any infection in the middle ear is present in conjunction with a so-called rheumatism, the offending organism can sometimes be isolated and an autogenous vaccine prepared.

Dr. Rogers said that his personal experiences, as regards the successful use of this form of therapeutics, had not been very encouraging, possibly because he had never been able to accurately differentiate the different forms of "polyarticular rheumatism"; for there is at least one of these acute or subacute joint diseases in which no bacterial contamination has been detected—i. e., the "rheumatism" which is characterized by the presence of the so-called Aschoff bodies in the heart, and which appears to be of metabolic rather than bacterial origin.

Vaccine therapy for polyarticular, nonsuppurative inflammation is thus a rather hit or miss treatment—at least, at present—but it is the best which we possess. It is indicated

only when there is discoverable some possible single focus of infection like an acute or chronic middle ear disease. Vaccine therapy is not indicated when no primary focus can be found, and hence the injection of the now popular so-called "phylacogen" mixture of organisms is seldom or never justifiable.

DR. EAGLETON said that personally he had had but one case in which the symptoms of joint involvement had overshadowed the ear condition. In all the other cases the ear was so obviously the cause of the infection that there was no doubt as to the diagnosis.

Dr. Oppenheimer had said that all the involvements of cerebral tissue were by direct continuity. Dr. Eagleton said that he had had one case which showed that this might not be so.

Dr. Eagleton said that the question of what to do for these joint involvements after a sinus thrombosis was one which had long interested him; years ago he had had an experience which had influenced his views on treatment ever since. The patient was a child who had complained of severe pain in its knee before the diagnosis of sinus thrombosis was made, but was operated upon for the thrombosis without paying any attention to the joint involvement. Two days later the pain became very severe, the temperature continued high, and it was discovered that one of the joints was involved, and an extension apparatus was employed. It was found that one knee had been dislocated; a week later the other hip became dislocated, and the child recovered with a permanently damaged hip. This case so impressed him that he made up his mind that in all cases of joint involvement, no matter whether it was thought to have fluctuation or not, he would aspirate the joint, and he has followed that procedure ever since. The hip is so situated that one cannot tell whether or not there is effusion in the joint. If the smear from the aspiration shows pyogenic bacteria, the joint is immediately opened and irrigated and the wound closed. This procedure has uniformly resulted in complete restoration of function. On the other hand, if there are no bacteria in the joint, many of these cases will recover if the joint is immobilized. The danger of delay, however, is very great, and every case of joint involvement associated with sinus thrombosis should be aspirated to find out whether or not it contains bacteria.

DR. LA FETRA: During the period from January 1, 1913, to November 15, 1914, there were treated in the children's wards of Bellevue Hospital one hundred and seventy-seven cases of otitis media. Of these, one hundred and thirty-two were acute, and for their treatment single paracentesis was done in thirty-six cases, double paracentesis in sixty-six—making a total of one hundred and two which were operated upon. There was spontaneous opening, frequently before the patient entered the hospital, in twenty-eight cases. The opening was double in ten instances and single in eighteen. There was no opening and no paracentesis done in eight cases. Among all these one hundred and thirty-two instances of acute otitis media there occurred the following complications: Acute colitis, one case; postauricular adenitis, one case; pneumonia, ten cases; sinus meningitis, one case; septicemia with pericarditis and pleuritis, one case. In the case of serous meningitis the blood culture was negative throughout, and the cells in the spinal fluid numbered on the fourth day four hundred and eighty; on the fifth day, two hundred and sixty-two; on the seventh day, twenty; and on the eighth day, thirty-seven. The infant made a complete recovery. In the case of septicemia with pericarditis and pleuritis there was acute purulent otitis media involving both ears. The infant was only seven months old, and died as the result of the general poisoning, the termination having been due to a hemorrhagic infarct in the right lung. It is to be noted that there was not one single case of mastoid disease developing in these one hundred and thirty-two acute cases. My personal experience in private practice has led me to feel that with early and perhaps frequent opening of the drum membrane, mastoid complications in infants and young children should be exceedingly infrequent. In the past fifteen years I have had arise in my own practice only five cases of mastoid complication. In many instances there was fear of mastoid disease, and of course mastoid tenderness was very frequently met. I have had no experience either in the hospital or in private practice with the complication of sinus thrombosis, and the hospital case mentioned of general septicemia is the only one I have seen.

During the same period there were chronic cases of otitis media to the number of forty-five. Double paracentesis was done in one of them and single in three. There was no open-

ing and no paracentesis in one instance, and there was spontaneous opening before entering the hospital in thirty-eight cases—double in twenty-two and single in sixteen. Among these forty-five chronic cases there was one instance of mastoiditis and one instance of temporosphenoidal abscess with no mastoid involvement. In this case the cerebrospinal fluid showed only five cells, but on account of the changes in the eye grounds, together with the general nervous symptoms, the diagnosis was made and the case was operated on at the New York Eye and Ear Infirmary.

Among the whole number of acute and chronic cases there was not a single instance of rheumatism or septic joints. It has seemed to me of interest to add in this discussion the number of cases of rheumatic fever and of sepsis treated in the hospital during the same period. During the years 1913-14 there were thirty-three cases of rheumatic fever. In one case the right drum was slightly congested and retracted; the left drum was normal. The ear symptoms were trifling and soon subsided. This patient had a hemolytic streptococcus blood; but at discharge this organism had disappeared and the drums were both normal.

During the same period there were treated in the wards thirty-three cases of valvular heart disease, some of which were acute, but most of which were chronic; one case of myocarditis and two cases of primary pericarditis. In none of these were there any symptoms or physical signs referable to the ear.

There were ten cases of septicemia during the same period. Of these, one infant of four months had acute purulent otitis on the right side, and one infant eighteen days old had a double otitis with meningitis as part of the general septicemia.

Altogether, then, there were among the one hundred and seventy-seven cases of otitis one case of septicemia, none of joint involvement. Among the thirty-three rheumatic cases there was only one of mild otitis; and among the ten cases of septicemia there were two with otitis.

DR. GUENTZER said that in connection with the subject under discussion, he would report a case that had been under his observation for a few months during the summer. The patient was a girl, eight years of age, whose adenoids and tonsils had been removed two years previously. On account of

repeated colds the family physician advised removal of remaining adenoid tissue, which was done on April 27, 1914.

On May 6th the patient came under Guntzer's observation, as she had complained of earache the previous day and during the night. The membrana tympani was bulging, and there was slight mastoid tenderness. A myringotomy was performed, followed by a free discharge for a few days. Culture showed streptococcus mucosus capsulatus. On May 11th, the tenderness having increased and the temperature being 102° , a mastoidectomy was performed at the hospital. The zygomatic cells were well developed, and the mastoid was filled with granulations. Again, the culture showed streptococcus mucosus capsulatus.

May 17th pus was evacuated from zygomatic region; for three days the temperature was fluctuating, and clinical symptoms of sinus thrombosis manifested themselves. Dr. John Richards saw the case, concurred in the diagnosis and advised jugular resection, which was done that evening.

May 20th, as there was no abatement of the septic temperature, the jugular bulb was explored, and pus was evacuated from that region. There was a distinct evening rise of temperature for nearly two weeks before a swelling of the right ankle joint appeared, with fluctuation. Both sides of joint region were incised (laterally) and drained.

On June 14th Dr. John Hartwell excised the astragalus, which was necrosed, and drained the joint through and through. On turning the child over, a bulging was found under the right scapular region. This was opened, pus evacuated; explored, and necrosis of wing of bone found.

On June 21st pus was evacuated from the right hip joint by Dr. Hartwell.

On July 11th Dr. Eugene Pool (for Dr. Hartwell, who had gone abroad) evacuated pus from behind the right hip joint and removed head of femur.

On July 15th the right femur was opened and pus evacuated from the medullary canal by Dr. Pool.

On August 12th there was a second opening and evacuation of pus from the right femur, below and behind.

During all this time the general condition of the patient varied. She had no chills, never vomited, and had no cerebral symptoms. She had paralysis of external rectus of left

eye, with some edema of the disc, and diplopia, which entirely cleared up in about three weeks. During these five months, pressure necrosis of the skin occurred wherever there was bodily contact.

The occiput was exposed, every spinous process of the vertebra, the crest of the pelvic bones, the posterior aspect of nearly all the ribs, the heels, and the contact surfaces of the bones of both legs.

Later the urine showed albumin and casts—hyalin and granular. Various blood counts showed leucocytosis and increased polynuclears. The warm days in the early part of September caused a watery diarrhea, from which the patient never rallied (either toxic or compensatory, from poor kidney action). The child died on September 26, 1914.

Dr. Guntzer said that this was one of the most trying cases that ever came under his observation.

Dr. Pisek agreed with Dr. Oppenheimer, that we do encounter arthritic inflammation following pus pockets or foci, whether in the sinuses, tooth roots, or elsewhere. As to the term "rheumatism," he did not like it, particularly in this connection. The complication was an arthritis, suppurative or nonsuppurative, and had nothing to do with the so-called rheumatic fever.

There is still in the minds of some practitioners the idea that a true "rheumatism" follows scarlet fever. There is a complicating arthritis in a certain number of cases, but rarely is it suppurative. The condition is one that is not yet generally understood, and it is to be hoped that light will soon be thrown upon it. We do see in young children a pneumococcic arthritis that is said to be primary. Pfister and Herzog had done some very good work along this line, without finding any focus elsewhere; there was probably a hidden focus which was not disclosed—hence the value of such papers as the one heard tonight.

From the standpoint of the infectious disease, it seemed interesting in this connection to present the number of cases of arthritis and otitis which occurred in the New York City Hospital for Infectious Diseases. In the diphtheria pavilion there were nine hundred and thirty-four cases. Of these, eighty-three, or 8.8 per cent, had at some time an otitis; among them no case of arthritis developed. In the scarlet fever

service there were one thousand and seventy-nine cases; two hundred and twenty-three had an otitis, i. e., 20.66 per cent. Among these were seventy-eight cases of arthritis—showing its more intimate connection with the streptococcus organism.

Collecting the cases for last year and this year, there were in all six thousand four hundred and eight cases of infectious diseases—scarlet fever, diphtheria, and measles—and of these six hundred and seventy-seven, or approximately ten per cent, had otitis. There were one hundred and twenty-eight cases of arthritis—or approximately two per cent; so that arthritis is not a common complication with scarlet fever, measles, and diphtheria cases.

The arthritis usually occurs in the second or third week of the disease, and when recognized and the joint immobilized it, as a rule, slowly subsides.

As Dr. La Fetra had said, in pediatric practice sinus thrombosis is comparatively uncommon in early life. Dr. Pisek himself had seen it in but one instance in the last three years, at the Willard Parker Hospital—a case that subsequently came to autopsy.

DR. MCKERNON said that the subject had been very well covered, and he congratulated Dr. Oppenheimer on bringing it before the attention of the section. There can be no question that metastatic abscesses do occur, and occur late following a long suppuration. Formerly it was not recognized that they occur so frequently as we now know is the case. Those who see these cases of septic sinus thrombosis very frequently ask themselves why they do not occur oftener. It has seemed to him that an explanation might be found in the frequency on account of the activity or virulence of the organism, and also in the resistance of the patients. Another point is that where these metastatic conditions developed late, it was very strange that they did not develop earlier, for in all the cases he had seen of metastatic abscesses subsequent to disease of the sigmoid sinuses, they had followed shortly after the acute process had developed or had been recognized. In one case it was some six weeks following the onset of the acute middle ear condition, and the acute process cleared up. The infection developed in the parotid gland; it broke down, and there was a sinus communicating with the external canal; and when the child was brought for examination, the hip was

anchylosed. The parents who brought it were told that the child required the attention of a general surgeon rather than of an aural surgeon. Later the abscess was opened and drained.

Dr. McKernon said that he was inclined to agree with Dr. Eagleton in reference to this kind of abscess, and believed that in every abscess of the joint following a suppurative sinus thrombosis, if examined carefully, a septic organism would be found present.

He again thanked Dr. Oppenheimer for bringing the matter to the attention of the section, for it was a very timely topic.

Dr. M. D. LEDERMAN said that a year ago he had presented before the section a child, seven years of age, showing a complication of this character, where the joint trouble appeared a week after the operation for resection of the jugular vein following mastoid and sinus disease. The child had a severe otitis and high temperature, and developed a pneumonic process. In the face of the active inflammatory condition of the lung, the child had to be operated upon on account of the increasing sepsis, characteristic fluctuating temperature, and tenderness along the jugular vein. Metastatic abscesses of both ankle joints developed, and bacterial examination of the pus showed the same organism as was found in the mastoid disease. Two inches of the vein were resected. The child made a good recovery, after having been ill for three months, with prolonged septic temperature. Antistreptococcus vaccine was also used.

In another case which had been presented before the section, the patient—a young girl, fifteen years of age, pregnant three months—had a chronic process of the middle ear with complications of the sinus and vein, followed by infection of the lungs. An abscess developed in the lung, but fortunately the contents of the abscess were evacuated through the mouth. The same organism was found in the pulmonary discharge, as was seen in the aural discharge. This patient also developed an alveolar abscess later on. Her symptoms on admission to the hospital greatly resembled typhoid fever.

In another case in which the internal jugular was ligated following mastoid and sinus infection, a child showed symptoms of joint involvement which did not go on to suppuration.

All these cases had occurred in the last five years, which would seem to indicate that the condition is not so uncommon as some of the previous speakers believed. Two of these children came to Lebanon Hospital within three weeks of each other.

DR. LEO B. MEYER, referring to the five cases of suppurative otitis media, with sinus thrombosis followed by pyemia, reported by Dr. Oppenheimer, said that it is not generally believed that such a joint suppuration could follow a suppurative otitis media without the intervention of sinus thrombosis. In one of the societies last year he presented an infant sixteen weeks old with suppuration of the knee joint. Though suppurative otitis media existed, the case was presented because of the marked sepsis and joint suppuration, with functional recovery of the joint. Dr. Oppenheimer had remarked that in ear suppurations when sepsis occurred, practically always a sinus thrombosis was also present. Dr. Meyer said that he was not familiar with the otologic aspects of the question, but on reference to the original article by Dr. Libman on bacteremia following otitis media, he found the statement that while he had never seen a case of general bacteremia following otitis media without the presence of a sinus thrombosis, and was not absolutely certain that this existed, he believed such cases had been reported in the literature. Especially in children did he believe this might occur. At any rate, this patient was an undoubted case of ear suppuration, with a suppuration of this particular joint and several metastatic abscesses, without a sign of sinus thrombosis, and the child recovered without an operation on its mastoid or sinus.

DR. SAYRE said that he had seen several cases of arthritis of the upper cervical vertebrae, following mastoid operations. These cases were frequently mistaken for torticollis, on account of the position in which the head was held, but they were undoubtedly metastatic infections of the second and third cervical vertebrae from suppurating ears, just as we see them from suppurating tonsils. One of these cases had been referred to him by Dr. Kerrison, who had thought it of sufficient interest to report to the society. Dr. Sayre said he did not know whether or not in this case there had been sinus thrombosis.

DR. MATTHEWS said that his work was with the ears and

not with the joints. He recognized the occurrence of these conditions of the middle ear and mastoid along with other general infections, but was not prepared to speak in regard to the percentage of their frequency.

DR. SPERRY said that he had had two or three cases that were similar to those that had been related by the other men. In one case of sinus thrombosis in a child six years of age, the child died with a multiple arthritis, and a large suppurating focus in the elbow joint. He expressed his enjoyment of the paper, and thought it was a very timely one.

DR. NORTON WILSON said that it was easy to see how the cases of metastatic involvement occurred when a thrombosis of the lateral sinus exists, but that it was not so easy to understand when there was only a simple otitis media. He had always felt that the latter class of cases must have gone through the lymphatics and involved the joints, or heart, or kidneys, just as it does by way of the tonsil.

NEW YORK ACADEMY OF MEDICINE,
SECTION ON OTOTOLOGY.

Meeting of January 8, 1915.

Tubercular Otitis.

DR. JOHN A. ROBINSON: Eighteen months ago the patient came to the New York Eye and Ear Infirmary, complaining of constant tinnitus in the left ear and slight deafness. Inspection showed a small yellowish spot at the tip of the malleus. No fluid in tympanic cavity. The patient had a tubercular lesion of both apices. He was kept under observation for four months, the spot gradually increasing in size until it was about one-eighth of an inch in diameter; then a spontaneous perforation occurred without pain. The tubercle broke down, leaving a fairly clean kidney shaped perforation. No tendency to granulation; no regeneration. Discharge scanty. Ear at times has been dry. Smear showed a mixed infection. No guinea pig inoculation made. The tinnitus ceased with the perforation.

DISCUSSION.

DR. HARRIS said that the case presented by Dr. Robinson was particularly interesting for several reasons. One was that attention was first directed to the condition by the tinnitus, and not by other symptoms. So far as pain is concerned, all recognize that it is not a characteristic of tubercular middle ear disease. He recalled one case, however, which showed a marked exception to this rule. The man was a patient of Dr. Forbes, whom he had seen in consultation some years ago and had a case of tuberculosis of the middle ear accompanied with the most excruciating pain. Dr. Harris said that he had never seen a case that suffered more, or in which he himself had suffered more because of the patient's suffering. It was an advanced case of pulmonary tuberculosis, and operation upon the ear was out of the question; the suffering was most profound and continuous, and extended over days and weeks, until all, including the patient, welcomed death.

It was the only case in his entire experience where there was such suffering in a case of tuberculosis of the middle ear and mastoid; but it would be well to keep it in mind as an illustration of the fact that pain can be present in tuberculosis of the ear.

An Obscure Case of Mastoiditis—Streptococcemia—Operation—Recovery.

DR. WILLIAM WESLEY CARTER: The following case from my service in Gouverneur Hospital seemed to me of sufficient interest to place on record:

A school girl, nine years of age, gave the following history on being admitted to the hospital on October 27th in the service of Dr. Francis Huber. Four days prior to admission she had been seized with a violent pain in the throat; this was accompanied by high fever and vomiting.

Examination at time of admission showed a follicular tonsillitis. Two days later the exudate on the tonsils, which at first was abundant, had almost disappeared. In the meantime the patient complained of pain in the right ear. This soon subsided without suppuration. The girl, however, seemed very ill; her prostration being altogether out of proportion to the severity of the tonsillar attack, and it was very evident that a complication had assumed first importance in the clinical picture. The patient at times was actively delirious, and the temperature ranged each day between 106° and normal; the time of highest temperature was 9:00 a. m. and 9:00 p. m., the lowest temperature being recorded at 1:00 p. m. The respiration and pulse kept pace with the temperature, the former ranging between 28 and 38 and the latter between 116 and 148.

On October 31st, four days after admission to the hospital, and eight days after the beginning of the attack, she was transferred to my service.

At 10:00 a. m., October 31st, the following clinical observations were made: Patient appears very ill indeed, is dull and apathetic. Temperature, 104° ; respirations, 28; pulse, 124.

Examination of Throat.—Pharynx congested, tonsils swollen, some exudation in crypts.

Ears.—Left, apparently normal. Right, slight redness and swelling of Shrapnel's membrane and extending along the pos-

terior wall of the canal. No perforation of the drum. There was no swelling over the mastoid, and no tenderness could be elicited by firm pressure over the bone.

Physical examination of the heart and lungs revealed nothing of importance.

The second metatarsal joint of the right little finger was swollen, red and shiny, and exquisitely sensitive. There were no other evidences of metastatic involvement.

The following reports had been received from the laboratory:

Urine cloudy; specific gravity 1015, neutral, trace of albumin, pus cells, granular casts.

Culture from throat negative for Klebs-Loeffler.

Blood examination for plasmodium, negative. Leucocytes, 22,500; polynuclears, seventy-eight per cent. Blood culture shows presence of streptococci.

Spinal puncture, fluid under normal pressure, clear. A culture of this showed subsequently that it was sterile.

In view of the above findings, the history of tonsillitis, the pronounced leucocytosis, the metastasis in the joint, the streptococcemia and the general appearance of the patient, we decided at once that her desperate condition was due to the streptococcic invasion of the blood stream.

The redness over Shrapnel's membrane and along the posterior superior canal wall pointed to the right mastoid as the probable source of the invasion. An immediate operation, therefore, was advised. As permission had to be obtained, this was not performed until 10:00 o'clock that night. At this time the patient's general condition was very bad and the outcome was considered doubtful.

A simple mastoid operation was performed. The antrum and the mastoid cells down to the tip were found filled with serosanguinous pus. The bony wall of the sinus was removed, but as the sinus appeared to be normal, it was decided not to open it, but to await early developments.

The day after the operation the clinical picture was entirely changed; the temperature dropped abruptly to normal and remained so. Recovery was prompt and convalescence uninterrupted. The metastatic joint subsided without supuration.

The patient was discharged from the hospital eleven days

after the operation, and the mastoid wound was completely healed a week later.

The features of the case to which your attention is especially directed are:

1. The desperate condition of the patient.
2. The slight objective signs of mastoiditis. (There being no tenderness over the mastoid.)
3. The metastatic involvement of the joint.
4. The pronounced leucocytosis and bacteremia.
5. The extensive involvement of the mastoid found at operation.
6. The fact that the sinus was not opened.
7. The rapid recovery of the patient.

DISCUSSION.

DR. PHILLIPS asked if Dr. Carter thought the case a purely sinus condition. It seemed to him that the tonsillitis would explain the involvement, rather than the ear.

DR. GUTTMAN asked what Dr. Carter meant by the expression serosanguinous pus.

DR. CARTER, replying to Dr. Phillips, said he thought it likely that the source of the metastasis was the tonsils, because when the patient came under observation the metastatic joint was very well developed—swollen, red, and shiny—and the child had been complaining of it for twenty-four hours before she was seen. The bacteremia may have been from the tonsillar infection, for there was nothing in the mastoid that pointed to invasion from that source into the sinus, for the sinus wall was apparently healthy. However, the tonsillitis had subsided at the time of the operation, and it seemed rather extraordinary that the temperature and all the other symptoms should so promptly subside after the operation, which would seem to lead to the belief that the infection of the blood was from the mastoid process. Whether the infection passed through the sinus, through the emissary vein, or through some other vein in close contact, it would, of course, be impossible to say. The extraordinary point was that the condition subsided so quickly after the mastoid was operated upon.

The term serosanguinous pus explained itself. It was a combination of serum, pus, and blood.

DR. HARRIS said that it was an extremely interesting subject, but if the case was going on record as a streptococcic blood infection cured by a simple mastoid operation, it should be clearly so understood. He would like to know from Dr. Dwyer, if in his wide experience it would be regarded as a unique case. According to the history, the temperature of the child dropped almost immediately after the operation.

DR. DWYER said that the unique point was that Dr. Carter went ahead and did the sinus and jugular operation. There seemed no doubt that the condition was due to the tonsillitis, and that the mastoid was a secondary condition lighting up and keeping up the symptoms.

All of these questions have been worked out very well by Rosenau simply by taking out the tonsils after an acute streptococcemia, or where the tonsils were very much infected; taking a culture from the capsules and injecting rabbits. He was able to reproduce the same culture as in the human being; and with rabbits he absolutely demonstrated the streptococcus in long chains, which is a parallel with Dr. Carter's case.

DR. PHILLIPS said that what he had really meant to infer was that it seemed to him that the operation on the mastoid had nothing whatever to do with the cessation of the symptoms. It seemed probable that the disease was taking care of itself and would have subsided had the mastoid not been opened. It was probably one of many such cases that all have seen which have a few symptoms and recover without any operation. It was difficult to understand an infection of a very serious type that would not produce more symptoms. The disease had probably reached its climax and would have recovered spontaneously.

DR. CARTER responded that the same might be said about any patient that got well after any operation. He had not especially emphasized the fact, but the patient was very ill indeed, and the parents were told that in all probability the child would not recover. That the recovery should have begun immediately after the operation was, to say the least, a very striking coincidence. He was convinced in his own mind—and it would require a good deal of substantial argument to change his opinion—that the patient's recovery was due to the removal of the infected mastoid cells.

DR. KOPETZKY said that he thought Dr. Carter was partly

right, and that he himself had just seen a patient who presented an almost parallel case. Dr. Carter's procedure had doubtless a great deal to do with the recovery of the patient. As he heard the history and compared it with the case he had just seen, it seemed that the case was one of septic sinus thrombosis; the streptococcemia was due to that; the type of mastoiditis which preceded the vein lesion was one which would bring a sinus thrombosis in its train. Dr. Carter opened the mastoid process and exposed the sinus and gave the lumen a chance to expand, and the one peculiar and particular factor which caused the formation of a thrombus was removed, namely, compression. It was not an obliterating thrombosis, and the patient recovered, owing to the relief of the pressure.

All are acquainted with Rosenau's work. You don't have to take streptococcus or staphylococcus from a tonsil. Any organism made into an emulsion and injected will show a predilection for joint involvement. Dr. Carter's case was a spontaneous cure of a parietal thrombosis by the removal of pressure on the outside. Dr. Körner had recorded a number of such cases, in some of which he had proceeded exactly as Dr. Carter had, and he, too, reported good results from this limited procedure.

DR. CARTER, in closing, expressed his gratification at Dr. Kopetzky's explanation of the condition, which seemed to him very probable. He said that he still believed that the recovery of the patient was due to the operation, and in view of the clinical history of the case, did not understand how it could be attributed to anything else.

A Case of Brain Abscess.

DR. FRANK T. HOPKINS: This case of brain abscess occurred in October at the New York Eye and Ear Infirmary, in the service of Dr. Dench, and came under my care.

The patient entered the hospital with a history of one month's ear trouble, but having had a discharge for only two weeks. A sagging canal wall and a tender mastoid showed the need of immediate operation.

The temperature was 100° (R.); the pulse, seventy-four to ninety. At operation but little free pus was found, but a perisinus abscess was disclosed near the knee. In removing the bone from this area the sinus was wounded.

A blood count and a blood culture were taken on the following day. The count gave 2200 white, with eighty per cent polynuclear. The culture was negative. The following day, i. e., two days after the operation, the temperature rose to 102.2° , while the pulse remained at ninety-four to one hundred. This was a possible beginning of the brain abscess.

This condition gradually improved until the fifth day, when the temperature was about normal and the pulse about eighty. For another two days the temperature was subnormal and the pulse sixty-eight to eighty.

On the ninth day, in the early morning, the temperature rose to 104° and later to 104.8° ; the pulse remaining ninety-two to one hundred. The blood count showed 6000 white, with seventy-nine per cent polynuclear. There was spontaneous nystagmus to the affected side—the second indication of brain abscess. There was cervical and occipital pain, but there was no stiffness of the neck. The Kernig was in question, and the Babinski toe reflex was lacking—or, rather, the toe remained stationary. It was not extended. There was no aphasia. The diagnosis, therefore, seemed a question between cerebellar abscess and sinus thrombosis. But the report on the following day on the blood culture showed a positive infection, and operation showed a clot in the sinus. The jugular was resected and the sinus was curetted until free bleeding occurred above and below the clotted portion. Three days later a blood culture showed no infection. The blood count was then 15,000 white, with seventy per cent polynuclear.

The temperature, however, remained 101° to 104° , with a pulse of from eight to ninety. The jugular wound and the sinus wound were exceedingly satisfactory. For five days after this there were no distinctive cerebral symptoms.

At the end of this interval, however, there was persistent occipital headache of intense character, and spontaneous nystagmus to the affected side. A general examination, made by Dr. Dench, disclosed nothing further. No reflex cerebral symptoms were found. (No vomiting, no Kernig, no Babinski, and no stiffness of the neck.) Cold water syringing of the affected ear diminished the nystagmus slightly and induced nystagmus of the opposite side. It was noticed that the pack-

ing towards the bulb was rather tightly placed, and this was made looser.

On the following day the patient was in every way better. The temperature was 99.5° to 100.5° ; the pulse, eighty to one hundred. There was no headache, and the nystagmus was exceedingly faint. During the next two days, in the absence of cerebral symptoms, an elevation of one degree of temperature was explained by a small collection of pus at the lower end of the jugular wound. On the twelfth day after the jugular operation, and one week after nystagmus was noted, there occurred, rather suddenly, all the symptoms of cerebellar abscess: nystagmus to the same side; occipital pain, intense and persistent; Kernig, Babinski; and the cerebellar reflex symptoms; pointing on the affected side was deflected to the same side. Diadokokinesis was present. The eye grounds, however, were negative. Spinal puncture gave a clear fluid, not under pressure. Examination of it showed Fehling and globulin tests, both plus, and the cell count to be ten. The cultivation was negative. Examination of the wound showed a good deal of pus in the sinus towards the bulb, absent the day before. This communicated with a cerebellar abscess of considerable extent. The abscess was opened and drained, but the patient died two days later.

DISCUSSION.

DR. PHILLIPS said that he wished to call attention to one symptom described by Dr. Hopkins, or to its absence—the absence of pain during the early stage of the disease. He also wished to emphasize another point that had appealed to him with great force in the last series of these cases that had come under his care—namely, that there may be a brain abscess of long standing and of considerable extent without producing any symptoms beyond headache. The fact that there is a brain abscess does not necessarily bring the symptoms one is supposed to find from reading the various textbooks.

DR. FRIESNER asked Dr. Hopkins how the cerebellar abscess was drained—whether through the inner wall of the sinus or from in front of the sinus, whether a counter incision was made into the abscess; also, how widely the dura was opened and how widely the abscess was opened.

DR. HOPKINS replied that the sinus was drained by an in-

cision on the inner side of the sinus where the abscess pointed, and it was quite thoroughly opened at that point. The condition remaining unimproved, the next day a counter opening was made behind the sinus; but the condition of the patient made it evident that there was no chance of recovery and that the second operation was not going to do any good.

DR. BYRNE asked what was the direction of the nystagmus, and also how the ear was irrigated, etc. In other words, what was done to eliminate labyrinthine disease as opposed to cerebellar abscess.

DR. HOPKINS replied that the nystagmus was toward the affected side. The syringing on that side produced nystagmus to the opposite side, and confirmatory tests were made.

The Isolation and Cultivation of the Tubercle Bacillus From the Discharging Ear in Cases of Chronic Purulent Otitis Media. (Preliminary Report.)

DR. G. H. COCKS AND DR. J. G. DWYER: Various methods have been employed in the past to diagnosticate aural tuberculosis, notably, von Pirquet's reaction, subcutaneous injections of tuberculin, animal inoculation, and the microscopic examination of tissue excised from the tympanum or mastoid.

Animal inoculation is attended with many difficulties and requires considerable time, during which the patient may disappear from observation. It is not always possible in chronic otitis to excise suitable tissue for microscopic examination. Subcutaneous injections of tuberculin and von Pirquet's test are open to the objection that the subject may be suffering from tuberculosis elsewhere in the body.

Our method for isolating and cultivating tubercle bacilli from the discharging ear appears to offer many advantages over the older methods hitherto employed.

Last year Dr. W. H. Haskin and one of us (Dwyer)* investigated the bacteriology of chronic purulent otitis media. The aural discharge was examined in all chronic purulent cases in which tuberculosis was suspected from the clinical signs or history. By the method pursued, the tubercle bacillus was demonstrated morphologically in six cases of otitis media and in one case of maxillary sinusitis. Reference is made in this report to some of these cases. The method used

*Haskin and Dwyer: New York Medical Journal, 1914.

by us was a modification of the antiformin method of examination of sputum, the modification being necessary on account of the difference in character of the aural discharge and sputum. The method was as follows:

The discharge was obtained in as large a bulk as possible in a small quantity of normal salt solution, the latter being used in amount just sufficient to wash out the pus. The water used in making up the salt solution was freshly distilled each day in order to be sure that none of the acid-fast organisms present in tap water or in old distilled water could vitiate our results. This discharge was then treated with an equal amount of fifteen per cent antiformin, and the whole was allowed to stand for a varying period, depending upon the consistency of the mixture, etc. It was then centrifugalized and the precipitate washed in order to remove the excess of alkali. Smears were then made from the precipitate and stained by Ziehl-Neelsen and Pappenheimer's method.

In this way we were able to demonstrate the organism with reasonable certainty. By this method, no matter how much care is exercised, there is always an element of uncertainty in morphologic diagnosis alone, as in many of the old chronic suppurating ears the acid-fast epithelioid flakes are apt to be mistaken for tubercle bacilli. These flakes are present in a large proportion of chronic cases.

In our own series, an effort was made to isolate the organisms from the discharge and cultivate them directly upon special media, so that there could be no question as to the diagnosis. Thus, animal inoculation was eliminated and much time was saved. This was made possible by Petroff's media, a full account of which appears in the *Journal of Experimental Medicine*, January, 1915. During the summer of 1914, Petroff, working in the Bacteriological Laboratory of the College of Physicians and Surgeons, Columbia University, devised a special medium which we have used successfully for cultivating the tubercle bacillus. Thus far we have tried this method on the discharge from thirty chronic suppurating ears, and have isolated tubercle bacilli in three cases. The three positive cases were found in making routine examinations of children who presented themselves at the Manhattan Eye, Ear and Throat Hospital suffering from chronic purulent otitis media.

The method we employ is somewhat different from Petroff's. It is really a combination of Petroff's method for isolating from sputum and his method for feces. This modification was rendered necessary by the large number of spore-forming organisms often present in chronic otitis. Our technic is as follows:

After obtaining the aural discharge in wide-mouth bottles it was immediately saturated with sodium chlorid and allowed to stand for one-half an hour to an hour. At the end of this time the bacteria were found floating on the surface. This floating film is then collected with a deflagration spoon in a wide-mouth bottle, and an equal amount of normal sodium hydroxid added. The mixture is shaken well and left for digestion in the incubator at 37° C. for one to two hours, or longer, care being taken to shake it every half hour. The mixture is then neutralized to sterile litmus paper with normal hydrochloric acid, and the sediment is inoculated into several test tubes. Growth usually occurs in from fifteen to thirty days.

At first no effort was made to differentiate the human and bovine types, but this is being done with the present specimens.

Nine cases of aural tuberculosis and one case of tuberculosis of the maxillary antrum were reported in the paper. Tubercle bacilli were isolated from the discharge in all ten cases.

DISCUSSION.

DR. HASKIN said that he had long been interested in the question of tuberculosis of the ear, and that several years before, he had presented one case before the Otological Society which he knew was tuberculous. He had tried in every possible way to find a means of staining it for tubercle bacillus, but failed, although he had found large masses of smegma bacilli. The first two cases in the Manhattan Eye, Ear, and Throat Hospital in which the tubercle bacillus was isolated were patients of his, and the discovery was made last winter when he and Dr. Dwyer were working together.

He was inclined to think that Dr. Cocks' estimate that one in every eight cases of chronic otorrhea in children was tubercular was a little too high. Thousands of cases are seen at

the hospital, and recently this condition has been sought for, and whenever a suspicious case is seen it is examined, and the bacillus is often found, but hardly as often as one in eight cases, if every chronic suppuration were to be examined.

DR. PHILLIPS said that he had been greatly interested in this series of experiments and had taken pleasure in forwarding them in every way he could, so that Dr. Cocks had had the opportunity of studying the secretions from a large number of patients. He further said that merely from a clinical standpoint he had reached a different conclusion from that expressed by Dr. Haskin, and for a long time had felt that a large proportion of chronic suppurative ears in infants and young children were tuberculous in origin. It had also been his experience that tuberculosis of the ear may occur without any symptoms of general tuberculosis, and may remain a local disease. At the Postgraduate Hospital on Monday he had seen a patient from the Raybrook Sanitarium with suppuration of the ear that had come on painlessly. The condition had been going on for a month or two, and examination of the ear showed distinct miliary tubercles on the drum. It must be remembered that children frequently have tuberculosis in different parts of the body—in the glands, the long bones, or the joints—and recover without becoming victims of general tuberculosis. We often see adult middle ears, now dry, which have formerly been the seat of extensive purulent processes, with the drum, the ossicles, and perhaps the outer wall of the attic all gone. This condition strongly suggests a tuberculous origin. About three weeks ago he had operated on a child who came to the clinic with a facial paralysis of one or two weeks' standing and a chronic discharge but no pain. There was an enlarged mastoid antrum from extension of pus, and granulation tissue. Specimens of the pus, granulation tissue, and bone had been sent to the laboratory, and he thought that Dr. Dwyer would be able to isolate the tubercle in this case also, for it seemed to be a clear case of tuberculosis.

DR. WILSON thought it would be interesting to know whether or not it was the bovine bacillus in these cases. It would look as though that might be a fact, inasmuch as so many of the patients were very young children.

DR. COCKS, replying to Dr. Haskin, said that of these thirty

cases, twenty-two were taken from a series at the Manhattan Hospital just as the children came in. It had been decided to test all the cases that came to the clinic with chronic sup-puration of the middle ear. The ears were washed out, and the discharge was sent to the laboratory for culture. Of the twenty-two cases, three were tuberculous—and this was the basis of his statement that in the experiments conducted, one in every eight of the chronic running ears was tuberculous. That was not a high estimate. He had not come to the meeting prepared to quote statistics, but for many years back there have been articles in the British journals by Milligan, Jobson, and Horne, one of whom had stated that in his opinion from fifty to seventy per cent of the cases of chronic otitis media are tuberculous. Another one stated that in his opinion, instead of this condition being largely due to the acute exanthemata, tuberculosis ranks in the first place, and the other infections come second. It was consequently very desirable in studying this subject to get accurate statistics regarding it.

DR. DWYER, replying to Dr. Wilson's inquiry, said that they had intended later to look into the question of whether the bacillus was of the bovine or the human type.

Paper: Accidental Injuries to the Sigmoid Sinus Occurring During Simple Mastoidectomy.*

By WILLIAM A. SCRUTON, M. D.,

NEW YORK.

DISCUSSION.

DR. HARRIS said that he had followed Dr. Scruton's paper with a great deal of interest, and that the doctor was to be congratulated on the very careful manner in which he had worked out the report. No doubt, every one would agree with his conclusions, and some of them might perhaps be emphasized. Instances of the wounding of the sinus during operation are far more numerous than some think, and though in the majority of the cases the results are not serious, yet there are probably many more serious consequences than have ever been reported. Certainly, all unnecessary exposure of the sinus should be avoided. Unless there are distinct indications to the contrary, it is good surgery not to lay the

*See page 310.

sinus open. Another point is that the landmarks are such that it is sometimes impossible to avoid wounding the sinus in a simple mastoid operation. Dr. Haskin had some specimens which would show that very clearly. Everyone knows that it is impossible to avoid wounding the sinus unless some unusual route is taken, such as in the Stacke operation. The most important point is that we must no longer regard it a routine procedure. Dr. Scruton had driven home with great force the fact that we are called upon whenever we open the sinus to exercise the greatest care to prevent infection from taking place. Undoubtedly, in some of these cases infection will occur in spite of every effort, but in others, by taking extra precautions, we may be able to avoid any serious complications.

DR. PAGE said that the paper was a very valuable one in that it called attention to the danger of an accident that was not generally regarded as very dangerous. Certainly, in the days when the antrum was entered directly with a chisel it was not an uncommon occurrence, and no record was kept of the consequences, but the operators seemed to regard it not as an error on their part, but as the will of God that the sinus was placed in the way of the chisel. As Dr. Harris said, it is a warning not to expose the sinus unnecessarily. There are times, however, when after the removal of the mastoid cells to get a cavity "like the inside of a tea cup," as Dr. Whiting expresses it, the inner plate is made so thin that its nutrition is likely to be impaired and quicker healing is gained from its removal over the normal sinus. Not infrequently a crack in it will necessitate its removal. Dr. Scruton's observations are sufficient warning to make him doubly careful from now on. He recalled a fatal case of sinus thrombosis which resulted from injury to the superior petrosal sinus during the removal of the cells from over it.

DR. KERRISON said that Dr. Scruton's paper was of undoubted value in calling attention to the possible dangers of a fairly common surgical accident. Only one detail in Dr. Scruton's method of treating such an accident seemed open to question. Dr. Scruton in each case stopped the hemorrhage with a gauze pad or plug, and before applying the final dressing irrigated the wound with an antiseptic solution. Dr. Kerrison believed that when the sinus has been accidentally

opened, it is safer to omit irrigations altogether. When the accident occurs, free bleeding should be allowed for a few moments, and then a plug of sterile iodoform gauze applied without great pressure to the site of the injury. This usually stops the bleeding promptly. If now we irrigate the wound, we do so only with the purpose of washing out infectious matter. But it is clear that at the same time infectious matter may be carried into or beneath the plug guarding the sinus injury, with possibly resulting intrasinus infection. It seems safer, therefore, in cases of accidental injuries, to simply cleanse the wound by wiping it out with sterile gauze or cotton.

Dr. Kerrison said that it was a distinctly courageous thing to bring before the section this series of cases. Those, however, who were personally acquainted with Dr. Scruton's work would know that the cases were treated with skill and care. The paper was, therefore, of decided value in calling attention to the fact that unintentional opening of the sinus is not without danger and is, therefore, an accident to be treated with respect.

Dr. BRAUN said that he was very glad to have heard Dr. Scruton's paper. He thought it a valuable contribution to otologic literature, in that it showed how far reaching and serious the consequences of accidental injury to the sinus may be. We have been inclined to look upon injury to the sinus only as an inconvenience in the completion of the operation, and have not thought much about the possibility of its leading to thrombosis.

He thought Dr. Scruton had been rather unfortunate in his experience. The large proportion of thrombosis following injury in his cases is not in keeping with the experience of most other otologists. He is convinced that this complication must be rather rare. He had never seen thrombosis follow injury to the sinus in any of his own cases, and only in one case operated upon by another man, in which the jugular bulb was injured.

The stage of the operation at which the injury occurs undoubtedly has some influence in determining whether thrombosis will result. If the injury occurs early in the operation, when the mastoid cells still contain large quantities of pus, there is certainly more likelihood of infection of the interior

of the sinus than when the injury occurs toward the end of the operation, when the mastoid wound is comparatively clean.

Injury to the emissary vein is much more common than injury to the sinus itself, but thrombosis following such injury is very rare. In every mastoid operation, veins are torn across which empty into the sinus and offer an opportunity for infection of the contents of the sinus. But in injury to the sinus wall there is an additional factor which favors the formation of a thrombus, namely, the roughening of the sinus wall by the injury. This favors clotting, and the clot serves as a culture medium for the bacteria in the mastoid wound. But there is still another factor which is of some importance in producing the thrombus. When the sinus wall is injured at operation, the sinus is compressed by means of a plug, in order to control the bleeding. This causes a marked slowing of the blood current in the sinus at this point, or even a complete stoppage, which fact favors the formation of a clot within the sinus.

There is undoubtedly some clot formation in a large number of cases in which the sinus is injured, but symptoms are present in only those cases in which the clot becomes infected.

DR. FRIESNER referred to the importance of the point made by Dr. Kerrison, and congratulated Dr. Scruton upon the interesting paper presented, but chiefly for his courage in reporting such serious and untoward results following technic error. Anyone who had watched Dr. Scruton operate would be confident that the mastoids had been thoroughly cleaned out.

One point which had impressed him especially while listening to the paper was that the results depend in a large measure upon where the sinus is injured. If it be injured somewhere through the mastoid cavity, before it bends forward to the bulb, it is comparatively easy to clean away the surrounding diseased bone. His own experience had been limited, and he could recall only one case in which he had injured the sinus in which any septic symptoms followed. In this case the injury occurred in the group of cells between the sinus behind and the bulb in front, underneath the facial nerve, and it was almost impossible to continue the operation and clean away the infected material in this narrow space. The patient had a single rise of temperature to 106°, followed by an equally

rapid fall, with uneventful and complete recovery. It was impossible to explain these phenomena in any other way than by the presence of septic material in the vicinity of the injured sinus which in some way got into the circulation.

As far as statistics are concerned, no conclusion could be drawn from Dr. Scruton's report.

In a report before the Austrian Otological Society, Rutin states that they operate on about six hundred mastoids a year. In these operations, the sinus is exposed from two hundred to three hundred times. They operate almost exclusively with the chisel and mallet, and we know that injury to the sinus is more frequent there than with us, though it would not seem that sinus thrombosis is appreciably more frequent with them than with us.

Dr. Scruton's experience was simply a piece of bad luck, and no one should conclude from it that injuries to the sinus are any more serious than has hitherto been believed.

DR. SCRUTON, in closing the discussion, said that the purpose of the paper was to make an initial record of cases of sepsis resulting from accidental injury of the sigmoid sinus, and to direct attention to the advisability of instituting measures tending to prevent infection of the repair process in these cases.

While iodoform gauze is an excellent styptic, and has definite antiseptic value as the result of liberating iodine when in contact with the tissues, it is not good drainage material; especially when prepared by the soap solution suspension method practiced in hospitals. Having this in mind, dressings in case number four were changed daily.

He expressed appreciation of the kind attention given to his paper.

Fracture of Skull—Hemorrhage From Meningeal Artery Through Middle Ear—Operation—Recovery.

DR. S. J. KOPETZKY: On the morning of Sunday, March 20, 1914, the patient left his house at eleven o'clock. Within fifteen minutes he returned in a dazed condition, undressed, and went to bed, where he shortly became unconscious and so remained until the Thursday following. During this period of unconsciousness he had no symptoms of anything in particular, excepting that he vomited for the first few days, the

vomit being tinged with blood. On Thursday, when he regained consciousness, he complained of pain in the right ear, and the attending physician called Dr. Kopetzky in consultation. When Dr. Kopetzky arrived, he found the patient in full possession of all his faculties, though slightly slow in cerebration. The ear drum had not been ruptured, but was somewhat bluish in color; and seen from the throat the eustachian tube gave evidence of having been bleeding. The tentative suggestion was made that the man had been hit on the head, or had fallen and sustained a fracture of the skull, and had bled through the eustachian tube, causing the blood stained vomit. The drum head was immediately incised to relieve the pain; no pus was obtained, but there was some bleeding, which continued from that time on for a few days.

The patient was removed to the Red Cross Hospital that afternoon for better observation, and that evening Dr. Strauss made a complete examination; lumbar puncture was then performed, and in the fluid old blood was found. From the neurologic aspect there was sufficient evidence to justify a diagnosis of fracture, with hemorrhage into the subdural spaces. The patient did well that night, and the following day (Friday) and Saturday, with no temperature elevation; but on Saturday evening he went into a state of coma from which he could not be aroused.

Meanwhile, an X-ray picture had been taken, which showed fracture parallel with the ridge toward the zygoma. Following the unconsciousness on Saturday evening, a decompression was performed at midnight. A flap was removed, as could be seen by the scars. Underneath the bone a tremendous amount of clot was found, which was carefully removed, and the dura was incised, the same way as the bone, but a quarter of an inch further inward, in order to give tissue to sew it up. One branch of the middle meningeal artery, by the posterior angle of the wound, was found to be still bleeding and spurting, one week after the injury. There was some difficulty in stopping the hemorrhage, but finally a piece of the temporal muscle was imbedded on the bleeding spot and sutured in place. The hemorrhage then stopped, the dura was sutured in place with fine stitches, and the patient made an uneventful recovery.

DISCUSSION.

DR. ARD said that a case of fracture of the skull was recently transferred to his service in Muhlenberg Hospital, on account of the ear symptoms. At that time he knew no other treatment than the expectant plan, and asked Dr. Sharp to see the case. Dr. Sharp did not advise operation in this case, but in others seen subsequently, where there was marked evidence of increased intracranial pressure, he advised subtemporal decompression. Examination of the eyes is very important in these cases, as the most trustworthy symptom of intracranial pressure is swelling of the optic disc, particularly its nasal half. Incision is made through the fibers of the temporal muscle, a section of the bone is removed, a crucial incision is made in the dura, and drainage with rubber tissue from the lower angle of the wound is made; the muscle is carefully sutured and a hernia of the brain is thus prevented. The same operation on the opposite side is done, if improvement is not effected by the first operation.

DR. FRIESNER asked Dr. Kopetzky if there were no external signs of fracture, no local edema or ecchymosis, and whether there was no paralysis on the opposite side, etc. Dr. Friesner said that he had recently seen a case where the patient fell in the subway and struck his chin, and returned home with practically the same history following as in Dr. Kopetzky's case. He had bilateral papilledema and Babinski, and no paralysis, although his coma continued for six hours. It was concluded that the middle meningeal or middle cerebral artery had not been injured, for if a clot had existed on the brain for five or six hours in the motor region, paresis or paralysis would have supervened.

DR. KOPETZKY, closing the discussion, said that the X-ray picture showed a linear fracture, whereas it had been supposed and was found to be a T-shaped one, an arm of the fracture running inward. The neurologic examination, made by Dr. Israel Strauss, had given ample data to justify the diagnosis.

Behind, when the flap was turned down, it looked exactly as flaps look when the two-stage operation is done—pasted down with clots of blood. The fact that the pressure was not continuous in the head, and that there was a vent for some

of the blood, probably saved the man's life. He was not in coma except for a short time at the beginning. Most surgeons will tell you that after a shock the patient will have a preliminary coma, followed by a secondary longer one. This man was only in the secondary unconsciousness for a very short time.

Dr. Kopetzky said that he was not trying to explain anything, but only stating the facts in the case as they knew them and as shown by the X-ray and the operation.

Acute Mastoiditis—Sinus Thrombosis—Simple Ligation of Jugular.

* DR. FRANCIS W. WHITE: L. S., age seventeen years. On October 29, 1914, this patient was operated upon for removal of a nasopharyngeal tumor, and was presented before the laryngological section last month by Dr. Harmon Smith.

At the time of the operation the hemorrhage was so severe that postnasal plugging was required, the packing remaining in place for forty-eight hours. A few days later pain occurred in the right ear, followed by a discharge. The discharge contained the streptococcus mucosus. One day later mastoid symptoms appeared, and the patient was transferred to Dr. Duel's service. The next day a simple mastoidectomy was performed. Pus was found most abundant directly over the lateral sinus, which was lightly covered by markedly necrotic bone. A culture of this pus showed the presence of the streptococcus mucosus. The temperature for the next few days was very irregular, being between normal and 101 to 102 degrees. A blood culture at this time was negative after twenty-four hours. An autogenous vaccine was administered on two successive days. The blood culture was negative after forty-eight hours.

On the ninth day after the simple mastoidectomy, and three days after the negative forty-eight hour blood culture report, an acute middle ear suppuration of the left side occurred, also a superficial abscess on a toe of the right foot, and one on the ring finger of the left hand, about the base of the nail. These were probably autoinfections due to picking of the parts by the patient. For the next two days the temperature varied between 99 and 103 degrees, and on the next two it went to 105 to 106 degrees, respectively. An X-ray of the left mas-

toid was indefinite. A blood culture showed the presence of the streptococcus mucosus the next day.

The lateral right sinus was opened, and for a moment there was no flow of blood, then suddenly it gushed forth from below upward. This was quickly controlled and the attention directed to the internal jugular vein. Several glands were of necessity removed, as they completely covered and were intimately connected with the carotid sheath. The vein was ligated below the facial, which subsequently was ligated. Neither vein was severed. Three days later a blood culture was negative. A swelling occurred on the external malleolus of the left ankle, which increased in size and redness, finally becoming bleb-like. A culture from the fluid showed the streptococcus mucosus present. Vaccine was given.

The highest temperature after the ligation of the veins was 104°, on the fourth day. Within two weeks after ligation the temperature was normal.

The highest pulse rate during the whole course of the disease was one hundred and twenty-eight; the lowest, sixty, on the day after the ligation.

The urine was negative throughout the disease.

Highest leucocyte count, 22,000; lowest, 12,000. The highest erythrocyte count, 2,200,000; lowest hemoglobin, seventy per cent.

The patient was in the ward six weeks. Since leaving the hospital, the patient has gained twenty-seven pounds.

A Brief Consideration of Some of the Factors Concerned in Cases of Atypical Sinus Thrombosis.

DR. SAMUEL J. KOPETZKY recalled a case which he had presented before the Otological Section on October 9, 1908, where there was found on autopsy a sinus thrombosis presenting in a mastoid process in which there were no cells present, and he recalled the fact that ever since that time he has been on the outlook for similar cases.

On November 30, 1913, he operated upon a male adult, aged forty-seven years, who after an attack of otitis media developed a sinus thrombosis without the intervening symptoms of an acute mastoiditis. The operative findings showed a similar lack or absence of all mastoid cells in the process. The case recovered.

Dr. Kopetzky then presented a number of anatomic specimens which he had collected, in which, while the outside contour conformed with a normal mastoid process, the interior was entirely taken up by the lateral sinus and the cerebellum. He contended that the infectious process, after involving the tympanic cavity, manifestly could not spread through mastoid cells which were absent, and so immediately attacked the venous blood channels. This constituted one form of atypical sinus thrombosis which had not received recognition to date.

Dr. Kopetzky then went into a discussion of other forms of sinus thrombosis atypical in one or other manners, and cited cases where the blood culture was positive, the sinus wall on examination found normal, and yet a thrombus was found in its interior, and he reviewed the operative findings from such cases, presenting the contention that these occur most often in those types of mastoiditis where the intercellular bony walls of the mastoid process are not broken down, best typified by the hemorrhagic type of mastoid infection. The appearance of the sinus wall, therefore, is no test in such cases as to the presence or absence of a thrombus. Finally, he cited cases where a similar type of mastoid infection was present, the blood and clinical picture one of thrombosis, and yet no thrombus demonstrable in the sinus; and he contended, in view of the bacteremia and the complications which some of his cases presented, such as secondary infection in the joints and muscles, that a thrombosis was present in the small veins in the walls between the mastoid cells; and he showed pathologic specimens of such thrombi. This group was another form to be classed as atypical thromboses.

The usual type of thrombosis with the discolored sinus wall and granulations on the sinus was found to occur mostly in the so-called coalescent type of mastoid infection.

The following charts summarize the paper:

CHART I.
Lesions Which Give Both the Clinical and the Blood Picture of Sinus Thrombosis.

Lesions.	Location.	Induced by.	Class of Case.
Phlebitis. Thrombophlebitis.	Lateral or sigmoid sinus. Dome of jugular.	Mastoid and tympanic infections.	Typical cases.
Thrombophlebitis.	Jugular bulb.	Tympanic and internal ear infections.	Atypical cases.
Osteothrombophlebitis or osteophlebitis.	Small veins in bony intercellular walls of mastoid.	Mastoid infections.	Atypical cases.
Osteophlebitis. Sinus thrombosis. Osteothrombophlebitis	Lateral sinus. Veins from labyrinth. Jugular bulb.	Tympanic or internal ear infections.	Atypical cases.

CHART II.
Typical Sinus Lesions.

Type of Tympanic and Mastoid Lesion.	Blood Vessel Reached Through Intermediary of.	Appearance of Sinus Wall.	Secondary Lesion.
Coalescent type.	Contact and erosion granulations.	1. Granulations. 2. Discolored. 3. Wall open into mastoid abscess.	Heart, lungs or none.
Hemorrhagic type.	Through thrombi of small veins in bony walls of mastoid cells.	Wall is usually normal in appearance.	Joints, muscles.
Chronic mastoiditis. 1. Bone lesion. 2. Cholesteatomata. 3. Acute exacerbations of O. M. P. C.	Contact of purulent tracts.	Rarely normal.	Heart, lungs. Intracranial lesions from thrombi and from original bone disease.

CHART III.
Atypical Sinus Lesions.

Type of Tympanic Infections.	Blood Vessel Reached Through Intermediary of.	Appearance of Sinus Wall.	Secondary Complications.
Acute middle ear infections.	Contact through floor of tympanum or small veins.	Normal.	Heart and lungs. Occasionally joints.
Acute middle ear infections.	Because of bone anomalies by contact or through network of veins.	Normal.	Joints and muscles.

DISCUSSION.

DR. BERENS congratulated Dr. Kopetzky on the complete and thorough manner in which he had presented the subject. The last word has not yet been heard on septicemia from ear disease, but we are learning, and learning rapidly, what Dr. Kopetzky endeavored to accentuate—that an osteophlebitis may occur in the mastoid and surrounding bone without any demonstrable clot or phlebitis in the lateral sinus. If Dr. Kopetzky had done nothing but accentuate that point and bring it home to the profession, he had accomplished a good work and his time would have been well spent.

All who had seen much of this work had seen such cases as Dr. Kopetzky had described—cases of septicemia with high up-and-down temperature. The sinus is uncovered, and the walls found to be healthy; it is opened, and no clot found. A complete exenteration of the mastoid and its cells is done, and the small thrombi are removed from the veins in the bone. These veins necessarily eventually enter into the general circulation, and we can get symptoms without really having a lateral sinus thrombosis.

Dr. Berens said that he had seen two cases where he went down into the innominate.

Dr. Berens said that he felt he must take exception to Dr. White's surgery. He had done a beautiful operation and the patient had made a beautiful recovery, and it might appear to be bad form to criticize the method employed, but where there is a clot present, the vein should be opened between the ligature and the clot, and drained at least, if it is not removed. In phlebitis where the vein is white or its walls striated, the vein should be excised as far as it is diseased.

DR. DUEL thought that the classification of cases of septic thrombosis which Dr. Kopetzky has presented was a very useful one. As Dr. Berens had said, it brought out very clearly what all have been gradually coming to realize—i. e., that in septicemia from mastoid and middle ear suppurations, the so-called atypical cases are as frequent as the so-called typical ones, if not more so. The time may come when we shall classify as typical cases certain cases of septicemia from aural suppuration which are now called atypical sinus thrombosis.

Dr. Duel agreed with Dr. Berens, that in many of the cases in which septicemia is present there is no demonstrable clot in the lateral sinus. For a long time he had found, nevertheless, that the surgery done for the relief of the situation—i. e., the isolation of the area which was taking in the sepsis—had cured the patient. His success in treating otitic septicemia had been much greater since he had gotten away from the notion that a clot must be exhibited in the lateral sinus or jugular bulb in order to demonstrate the necessity for operation. The question of whether or not in the presence of a clot in the lateral sinus the surgery suggested by Dr. Berens is the best, is one which presents opportunity for much discussion. Personally, of late years he had found very few cases where he thought it necessary to resect the jugular vein, although he had formerly always adopted that method. At present, as a rule, he simply ties and severs it from above the facial vein unless, after exposure, he finds that the clot has extended beyond that level.

Within the cranium he plugs the sinus toward the torcular, well beyond the clot. The sinus is split and laid open from this plug to the bulb. If the thrombus is easily removable without great hemorrhage, it is done at once, but in most cases, to avoid the dangerous hemorrhage, he is usually satisfied to remove the offending clot at the first or second dressing following the operation, if he has been successful in stopping the clinical evidence of general sepsis for which the operation was performed. The presence of the thrombus is locally not a great menace. To spend much time trying to remove it after the circulation is cut off at the torcular and jugular ends is unwise, unless the petrosals are plugged, as the loss of blood is appalling at the time, and much more dangerous for the patient than the now localized septic clot. One to three days later, the clot can be removed without hemorrhage—the petrosals having been plugged off by a healthy clot as soon as they were unable to deliver blood into the sigmoid and bulb.

DR. BERENS explained that he had not intended to be understood to say that he exsects the jugular from the clavicle up. If that impression had been given, he wished to correct it.

DR. KERRISON said that apparently a useful deduction might

be drawn from this paper in determining the surgical indications in certain cases. Dr. Kopetzky's report seemed to show so many avenues of infection from a diseased mastoid to the jugular system of veins, that in cases of suspected sinus infection it would seem safer to ligate or resect the jugular, even though no physical evidences of a clot or septic lesion could be demonstrated in the sigmoid sinus or bulb.

Dr. CHAMBERS told of an atypical case in which the septicemia had continued for a month after the radical operation had been done, and it had seemed as if it must be a case of sinus thrombosis, although there were no symptoms excepting those of septicemia. Dr. Kopetzky had seen the case, and advised opening the vein. The patient had the septicemic temperature curve for certainly a month afterward; then he had a perirenal abscess, which was opened two or three months ago, and recently he had returned with a second abscess in that region; an inquiry about the condition of the ear elicited the response that that was all right.

Dr. HELD said that Dr. Kopetzky's paper had brought out a number of excellent points, and had also reminded those who keep records of our operative findings that we all have had experience with a number of these cases of atypical sinus thrombosis. After speaking with Dr. Kopetzky on this subject a few weeks previously, he had looked up his records and had found more of them than he had thought. One particularly interesting case was typical in every way, and yet the operative findings had showed nothing—but the plugging off of the sinus resulted in immediate cure. We should all remember that when there are clinical symptoms of septicemia with suspected sinus thrombosis, we should follow the same treatment that we would if we knew there was a clot—uncover, ligate, and remove clot if found.

Dr. IMPERATORI said that Dr. Kopetzky had omitted, in his classification of atypical sinus thrombosis, to mention one type of case, that is, those instances in which the middle ear was not infected, occurring in scarlet fever with cervical adenitis. The internal jugular vein becomes thrombosed, and there is an extension of the process to the bulb and sinus. Several such cases had been reported before the section.

Dr. HELLER said that these cases very often tax the judg-

ment as to just what should be done, and where to stop. A month ago Dr. Carter had reported a case of a child with septic symptoms, who recovered after a simple mastoid operation. If we are to stand on the footing as reported this evening, are we always—when we have an apparent or true septicemia and find that the vein looks normal—to tie off the jugular vein? When are we going to choose? Dr. Carter had shown that a simple mastoid operation in a case of undoubted septicemia, cured the patient. The question is, What one sign or group of signs can we take as a standard as to when we should go in and open, and when to tie off the vein? We do not want to subject the patient to a needless operation if it can be avoided, and we do not want to open them all.

DR. WHITE said that he had nothing to add to the discussion excepting to say that he was following the teaching of the Viennese school—where ligation of the internal jugular was practiced, but not its removal—the results of which up to the present time have been exceedingly satisfactory. As a matter of fact, he had not seen the removal of the internal jugular vein for lateral sinus thrombosis until he became a member of the visiting staff of the Manhattan Eye, Ear and Throat Hospital.

DR. CARTER said that Dr. Kopetzky had brought up a most important subject just at this time, and it should be remembered that venous stasis, venous thrombosis, and exudation of blood corpuscles through the walls of the vessels form part of the inflammatory process—is really the latter part of the third stage of inflammation; and while that is normally inflammation, it should be borne in mind that it has this important bearing upon sinus thrombosis. The case which Dr. Carter had presented at the last meeting of the section and which had been mentioned by Dr. Kopetzky and others in the discussion, illustrated admirably the principles emphasized in the paper of the evening.

DR. WHITE asked if Dr. Kopetzky had had any experience with the vaccines, of which so much is heard nowadays, and said that he had many offers from various vaccine enthusiasts to cure cases of mastoiditis that came under his care.

DR. GUTTMAN said that he had been much interested in the paper, but that there seemed to be some misunderstanding in

regard to the clinical picture which Koerner describes as osteophlebitis. Koerner considers osteophlebitis not necessarily in connection with sinus thrombosis, but a disease by itself, with well defined clinical symptoms. Osteophlebitis is caused by an infectious thrombosis of the small veins in the mastoid bone in cases of acute middle ear suppuration with or without mastoiditis. As a rule, these cases get well without surgical interference.

We occasionally see some cases of atypical sinus thrombosis. Only a few weeks ago he had such a case under observation. A child, about four years old, was suffering from acute purulent otitis media of the left ear, when one day he began to complain of the right ear. Dr. Guttman was called in and made a paracentesis on the right drum membrane, after which the child felt quite well for a few days, and then suddenly became very seriously sick, with chills, vomiting, semi-consciousness, and a temperature of 106° . The temperature varied between 104° and 106° . Locally, there was noticeable only a slight edema over the left mastoid bone. Dr. Guttman was prepared to open the antrum and lay bare the sinus, when, thirty-six hours after the onset of the disease, the temperature went down and ultimately became normal. The next day the pulse of the child became very irregular, which suggested the thought of a septic endocarditis. This symptom also subsided after twenty-four hours, and the child left the hospital perfectly well, after a stay of four days.

Had this child been operated upon, the sinus opened and no thrombus found, it would have been considered one of the atypical cases of sinus thrombosis. It is true, one cannot always rely upon such a fortunate outcome. Every case must be judged individually, and there will always be found cases which cannot be classified in any of the groups presented tonight.

DR. JOSEPH BYRNE said he had not been able to orient himself clearly on the case, as stated. He understood Dr. Koptzky to say there was a linear fracture of the bone discovered parallel to the zygomatic arch at such a height that it could not possibly have involved the petrous bone, and as there was no mention of a fracture of the base, or complete rupture of the dura over the convexity, he did not see how

blood could have reached the tympanic cavity. The cerebral dura mater consists of two layers over the convexity. The outer layer, rich in blood vessels and nerves, acts as periosteum for the inner surface of the cranial bones, whilst the less vascular inner layer, consisting of more or less cellular fibro-elastic tissue lined on the cerebral surface by a single layer of flat cells, constitutes the investing membrane proper of the brain. Between these two layers of the dura are found large venous sinuses. In fractures of the vault the outer layer of the dura is frequently injured without rupture of the inner cerebral layer. This gives us the epidural variety of hemorrhage, with its well known sequence of clinical phenomena, e. g., history of cranial injury, immediate temporary unconsciousness due to concussion, followed by a period of lucidity, which in turn, as the arterial hemorrhage progresses, gives place to the persistent progressive coma of progressively increasing intracranial pressure. Over the base the dura forms a single compact membrane closely adherent to the bone. In this region it is practically impossible to have fracture of the bone without rupture of the dura. Hence fractures of the base are usually of the compound variety, and the hemorrhage resulting therefrom is always of the subdural variety.

Now, as lumbar puncture had shown blood in the cerebro-spinal fluid, the conclusion is inevitable that there existed subdural hemorrhage, and as there was no mention of the inner layer of dura having been found torn at the operation done for epidural hemorrhage, it seemed we were here dealing with a fracture of the base through the petrous bone. In no other way could blood reach the tympanic cavity. But perhaps the neurologic examination (which was not read by Dr. Kopetzky) may throw some light on the matter.

DR. KOPETZKY thanked the gentlemen who had taken part in the discussion, and apologized to Dr. Imperatori in that he had limited his presentation to cases of sinus thrombosis of otitic origin.

Replying to Dr. White, he said that he has only a limited experience with vaccine treatment. The more serious cases he had reported, and some of which recovered, had not been given vaccines. The second case to which he had referred had received Hiss extract, and had recovered, but he was not

inclined to ascribe the cure to the use of the extract. He could not give a more definite statement on the vaccine question. To his mind, their value remained as yet unproven.

It was very difficult to analyze the case spoken of by Dr. Guttman. Some cases properly treated, die, while others improperly treated, get well. Dr. Kopetzky said he would have ligated in this case rather than take the chances which Dr. Guttman had taken.

Paper: A Simple Manometric Apparatus for Measuring Eustachian Tubal Patency and Tympanic Ossicular Mobility.*

By L. M. HUBBY, M. D.,

NEW YORK.

*See page 307.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, November 17, 1914.

DR. OTTO J. STEIN, THE PRESIDENT, IN THE CHAIR.

Case of Brain Abscess.

DR. OTTO J. STEIN showed the specimen from a case of brain abscess. The patient was a young man of twenty-six, who gave a history of having had chronic suppurative trouble with the middle ear for nineteen years, with occasional acute exacerbations, without any unusual symptoms. His last attack occurred in July. He was seen three weeks after the onset of this acute attack, and presented the following history and appearance: Family and personal histories negative. Married; one healthy child. Habits good. When he entered the hospital he showed nothing particular, except that he was suffering very severely from a headache of the type suggestive of some intracranial complication. There had been considerable discharge from the right ear, which had practically stopped the day before entrance to the hospital. The severe pain developed a day or two before coming to the hospital, and continued to grow worse. He became nauseated, and vomited all the night before and morning of entering the hospital. The mentality was perfectly normal. Temperature about 100.5°. That evening it dropped to normal, and remained within a range of a degree and a half. The pulse was rather striking—fifty-six to sixty-four—not very full, but rather weak, and not very regular. The smear from the ear showed a mixed infection of pneumococci and staphylococci, with a few short chains of streptococci. X-ray pictures taken the next morning showed nothing, so far as the ear was concerned, from the speaker's standpoint, but others who saw them thought they gave positive findings. The blood count showed 19,000 whites, with an eighty-five per cent polynuclear count. Urine normal. There were no particular eye symptoms; no nystagmus; no irregularity of the pupils; they re-

acted a little slowly to both light and accommodation, and were equal.

The points he wished to emphasize were: Temperature about normal, subnormal pulse, severe head pain and vomiting.

He diagnosed a mastoid, with a probable brain complication. Operation showed the sinus lying very far forward, presenting immediately with the first chip of the chisel. The middle cerebral fossa also lay very superficially. This made it very difficult to find the antrum, but the entire bone was involved, as in an acute mastoid. The only pus found was in the posterior cells of the zygoma, just around the floor of the middle fossa, and there five or six drops were found, in which the organisms above mentioned were found. The dura was dark red, thick, fleshy-like, about the area of the middle finger nail. It was not adherent, and no extradural abscess was found. Then the sinus was uncovered and showed no trouble. The antrum was found with difficulty; it was full of granulations and pus, the smears showing the same organisms. The speaker then decided to do a radical operation. He took out the bony posterior wall, and left the wound open, without opening the dura.

The next morning the man was practically free from pain, the other symptoms having also subsided. The temperature was normal. That afternoon at about four o'clock the same condition prevailed, but in the evening he was called up on account of the return of the severe pain; temperature went up that night to 102.4° ; nauseated; there was a great deal of gas in the stomach and intestines. Breath very foul. Tongue badly coated. The pulse kept getting slower again, from seventy to fifty-four. Mentality not clouded.

Unfortunately, Dr. Stein had arranged to go away about this time, but hesitated about leaving this patient, without opening the brain, so decided to operate again. He enlarged the opening formerly made around the posterior root of the zygoma—just at the bend—and found healthy dura. Then he passed the knife through the dura and into the brain in the temporosphenoidal area, back as far as anyone would want to, and could find no pus. He then pushed the knife upward, and found nothing; then anteriorly, and found no pus. He was rather chagrined, and put a small drain in—a gutta percha

cigarette drain. The next morning the man was perfectly free from pain and felt comfortable; mentality clear; temperature had dropped to 100° , but the slow pulse still persisted.

Dr. Stein left the city the next day, leaving this patient in the care of Dr. Fletcher and Dr. Jones. During that week the patient's condition fluctuated. A lumbar puncture had been done at the operation, and a very decided pressure found; the same organisms were found in the fluid as in the former smears made. There was also a little blood present in the fluid. A second lumbar puncture was made during Dr. Stein's absence—four or five days after the operation—on account of severe symptoms. The puncture relieved him. Fifteen cubic centimeters of fluid were removed. From that time he seemed to progress very well; however, he always had a little headache and a little temperature. During the following week it never rose above 100.5° . The pulse improved, as well as the general condition. He was apparently on the way to recovery. When Dr. Stein returned he was quite confident of the man's recovery; only now and then he would complain of lancinating, sharp pain in the head. Three or four days later he suddenly got much worse; the pain in the head became very severe; the nausea returned, and symptoms of a severe nature developed, so it was thought another puncture might be a good thing. No indications seemed to be present for opening the sinus. On puncture there was no pressure; but the fluid was a little cloudy. There were no organisms, but some pus cells. This puncture gave him no relief whatever. He continued to get worse until all the classic symptoms of the terminal stage of abscess developed, and he died—about two weeks after he was first seen.

On postmortem an enormous abscess was found in the right temporosphenoidal lobe, with hardly any capsule. The same organisms were found.

The question in Dr. Stein's mind is: While it is easy enough to make a diagnosis of brain abscess with these symptoms, and sometimes even to localize the same, and even to get the consent of the family to operate, still the question is to know exactly when to open into a brain. His idea is that this patient, at the time of operation, was developing a brain abscess; the brain was in a state of red softening. He does not think he infected the brain. He believes he operated too soon. If

he had operated later—say, on his return to the city, and at the time the last lumbar puncture was made—he probably could have opened the abscess and drained it, with recovery of the patient. However, he feels that instead of tapping the spine for the third time he should have sought the abscess, and it no doubt would have been found then. That, he thinks, is the most important thing about the case, and he wished to hear the experience of the other members in regard to the time of opening a brain abscess.

DISCUSSION.

DR. HARRY KAHN asked what was the location of the abscess.

DR. STEIN said it was in the temporosphenoidal lobe.

DR. J. R. FLETCHER recognized the case. In answer to Dr. Stein's query as to when to operate in these cases, he would say that he would be very much inclined to open these abscesses as soon as he could make a diagnosis. In the case reported, the speaker was called to the hospital during Dr. Stein's absence from the city, because the man had developed a state of delirium. The blood pressure had increased very much. For this reason another lumbar puncture had been performed, after which he improved so much that it was thought he would recover. Right after that the urine was examined, and the organisms referred to by Dr. Stein were found present in large quantities. The termination in this case was a great surprise to Dr. Fletcher, because the man was apparently recovering so nicely, and even volunteered the information to those around him that he was going to get well promptly. But as to the time of operation, he would operate when he thought he could make the diagnosis of brain abscess. His own idea is that the abscess was present, but small, when Dr. Stein operated, and therefore the knife blade did not reach it.

DR. GOOD asked in what way the increased pressure was determined.

DR. STEIN replied that it was determined by the appearance as the fluid came out.

DR. FLETCHER said that they took the blood pressure while Dr. Stein was away.

DR. JOSEPH C. BECK was very much astonished that Dr.

Stein neglected to follow the modern method of treatment of brain abscess, which is to open the dura and wall off the area, and let the abscess, if it will, progress to the surface; then open in the second stage, about a week or ten days after. That is the preferred method of treating brain abscess at this time, as shown in a series of cases operated by McKernon, Dench, and others, that that method gives the best prognosis. While his (Beck's) results up to this time have been very poor in the treatment of brain abscess, with this method he has had the best results. Therefore, he would say, contrary to Dr. Fletcher's statement, that in this case the dura should have been opened if the diagnosis was made of abscess the second time patient was operated upon; the tendency would have been for the abscess to form a walling off, and then incision and drainage would have probably given a better result, in that the healthy brain tissue was not infected.

DR. J. R. FLETCHER said he was not willing to accept any man's method as a modern method. All methods are modern methods. Simply because one man makes a statement that this, that or the other is better, he is not prepared to call that a modern method, or be surprised that one does not follow it. He would operate on a brain abscess, even now, just as soon as he made the diagnosis.

DR. BECK said he would operate immediately, but in two stages.

DR. FLETCHER said he would open the abscess if he could find it.

DR. BECK said that Dr. Stein should have waited until the abscess had had a chance to come to the surface.

DR. STEIN asked Dr. Beck if he meant that he should have let an exudate form there, to which Dr. Beck replied in the affirmative.

DR. STEIN said that he had presented the specimen and described the case in order to get the opinions of the members. He was glad to hear that Dr. Fletcher has a definite idea as to what he would do with a case of brain abscess. He would admit that he had never walled one off. He has opened them, in cases where he has found the abscess, and has not had bad results. Others probably have had the same experience as he. Of course, he has had fatal results in cases where he has

found the abscess, also, even if well walled off. It was interesting to know the attitude of the profession, and it was probably also of value.

Paper: Ethmoidal Exenteration.*

BY CHARLES M. ROBERTSON, M. D.,

CHICAGO.

DISCUSSION.

DR. O. T. FREER said that the entrance into the ethmoidal labyrinth through the bulla, suggested by Dr. Robertson, a way also advised by Boenninghaus and Vacher, is a good one, and often better than the route through the agger cell of Mosher, for this cell is sometimes absent or unduly thick-walled, while the bulla is frail and easy to see when the middle turbinate has been resected.

The chief characteristic of chronic ethmoiditis is the formation of polypi. When superficial, they do not affect the skeleton of the ethmoid region. In the common deep type of ethmoiditis, however, where the polypi form within the cells, they destroy the cell walls by pressure absorption, and the entire lateral mass of ethmoid cells usually degenerates into a polypoid mass, with occasional thin plates of bone remaining as vestiges of the cell walls. The degeneration virtually dissects out the entire ethmoid mass of cells, including the frontal cells of Killian, which form the floor of the frontal sinus, so that a probe or curette enters the widely open sinus in the course of the operation. In such cases the bulla, the agger cell, the processus uncinatus, the middle turbinated body, all of the landmarks, are gone, and the operator has to trust to the mental picture of the ethmoid region anatomic study has given him, and to his fine sense of touch, which tells him when his curette has gone beyond the region of degeneration and has reached healthy, hard bone. This bone, in the outward direction, may be the firm walls of sound ethmoid cells, or else the degeneration may have reached the lamina papyracea of the orbit. Considering the frailty of this plate, the greatest caution is necessary in approaching it, and Dr. Freer had been told of two cases where it had been penetrated.

*See page 319.

Very gentle management of the curette and punch is needed to insure safety.

Upward, the curette may have to travel to the tegmen or cranial roof of the lateral mass of ethmoid cells. The cribriform plate is not in danger if the curette be kept outside of the line of the vertical plate that bounds the ethmoid cells inwardly and ends in the middle turbinate below, as shown by P. Watson Williams (*Journal of Laryngology*, May, 1914).

Where no polypoid degeneration of the ethmoidal labyrinth exists, Mosher's landmarks and the bulla are easily found.

Resection of the perpendicular plate of the ethmoid bone in the nasal septum to the cribriform plate, as suggested by Dr. Robertson, Dr. Freer thought dangerous. The perpendicular plate often has to be cut away to within one-half inch of the cribriform plate, but to remove it up to the latter would involve a chance of meningitis, because of the intimate lymphatic connection between the nasal lining and the dura in this region.

DR. H. W. LOEB, of St. Louis, speaking with reference to the cells mentioned by Dr. Robertson as being found in the middle turbinate, was not willing to accept them as always being due to inflammatory processes. The interesting thing about them is that the opening is almost always near the roof of the cells, rather than near the floor, as in the other ethmoid cells.

He reported an operation for extensive disease of the ethmoid, in which he unfortunately entered the periorbita. He took out several pieces of what he thought were strange-looking polypi, and found that they floated, which showed him that it was periorbital fat he was removing. In this case, if there was any hard bone on the orbital side of the ethmoid cells, he did not strike it. Either the disease had destroyed it, or there was a dehiscence. The patient had a very violent reaction, but recovered.

DR. R. H. GOOD said, in regard to saving the middle turbinate body, that he tried faithfully to carry out this principle some years ago. However, he has found that many of the cases are polypoid degenerations; there are many in which the upper nasal space is very narrow, and many in which the middle turbinate, on account of enlargement or degeneration, needs to be removed. If it is not removed, at least the anterior por-

tion, in some of these cases it causes considerable disturbance afterwards. Especially has he noticed this in the intranasal frontal sinus operations, where he would leave the middle turbinate in place. There would be considerable congestion in the nose, and the patient would not get along nearly so well as if the anterior turbinated body and ethmoidal cells had been removed.

In regard to cleaning out the ethmoid cells, he has done quite a number of these operations. He goes through the bulla and bites an opening upwards and backwards to the base of the skull with forceps. Then he takes the frontal sinus rasp, with the smooth surface toward the orbit, beginning in the posterior ethmoid cells, coming forward and rasping out the ethmoid cells up into the frontal sinus. He considers this method very simple and thorough.

DR. ROBERTSON, in closing, referred to Dr. Freer's remark that the bulla is sometimes destroyed, so that we are unable to find it. Dr. Freer can easily find the bulla, and so could the speaker, and so could all of the members, by the anatomic relations in the nose—even with eyes shut. Dr. Freer knows exactly where it is, at what distance, and at just what angle. So the speaker felt sure Dr. Freer would retract that statement.

DR. FREER said that he had previously stated that the bulla is frequently so diseased that it is lost. Of course, if it is present he could find it.

DR. ROBERTSON, again referring to Dr. Freer's remarks, said that Dr. Freer had said "that to preserve the turbinate would be to preserve pathologic tissue." The speaker had said that these were cases in which the case had been operated first, and that pathologic tissue had already been removed. There are cases of this type in which all of the tissues of the nose have degenerated, and sometimes we find noses in which there is a myxomatous degeneration of the entire contents of the nares. In such a case he would not pretend to do the type of operation described.

The rule of going into the nostril until you find a hard lamina of bone is a dangerous one to pursue unless you go anatomically, because the lamina papyracea is often dehiscent, as Dr. Loeb said, and one is liable to get globules of orbital fat. He has seen cases in which the lamina papyracea was so

thick that a steel curette would not go through it. Individual peculiarity comes into account in these cases.

He would not attempt to remove the septum as far as the cribriform plate. He would expect to remove it as far as possible toward the cribriform plate, however. There is a danger of meningitis, just as there is a danger of meningitis in any operation on the nose. Of course, the nearer you get toward the cribriform plate, the more you subject your patient to risk.

Dr. Loeb said that "these cells were not always due to an inflammatory process." Dr. Robertson did not say that they were, but he thinks that there are some of them that are. In examining skulls, one very often finds large cavities in the turbinates, both in the middle and inferior. Where there is much disease, the middle turbinate is still pathologic. It is pathologic, but you save it with the idea that it will at least do some of its function after the disease has settled down. If it does not, you can easily take it out afterward, because it is a painless operation, and only takes a short time. After the cells have been removed and the turbinate fractured, there is still some space where the cells were, so that if there is a continuation of pus formation, it can easily be mopped out.

Dr. Good said "he had tried to save the middle turbinate, but that it was inflamed." Surely he would not expect to take out an inflamed turbinate at any time. If treated properly, the inflammation will subside. This can be done to a considerable extent by medication in the nose. Unless in a pathologic condition, you would not expect to take it out at all.

"The space is too narrow in some of these cases." That is just the object of the operation described, namely, to get more space and still save the turbinate. You can get at least one-eighth inch in the transverse diameter in the nose by this method, and that is plenty for ventilation.

"In many of these cases there are polypi." These are polypoid and do not constitute a diseased condition in a case of that kind; they simply are a symptom of pus, and after the pus is relieved the polypoid tissue will disappear. If the polypoid tissue persists, you may be sure that you have not reached the ultimate end of the ethmoid cells that produces the secretion which results in the polypoid tissue.

Paper: The Use of Pituitary Extract as a Coagulant in Surgery of the Nose and Throat.*

BY HARRY KAHN, M. D., AND L. E. GORDON, M. D.,

CHICAGO.

DISCUSSION.

DR. L. E. GORDON said the principal point that they wished to bring forth was that the extract of the posterior portion of the hypophysis, when injected, influences the coagulation of blood by decreasing its coagulation time. The coagulation time is decreased within fifteen minutes, and it remains so for over twenty-four hours. What causes this coagulation? Francini, in 1910, while working on metabolism with pituitrin, found a lessened amount of calcium in the body tissues, and a greater amount of calcium thrown out into the blood stream. This point may be worth further consideration.

The injection of pituitrin has been found to practically stop bleeding in almost every case. A great deal more work can be done with pituitrin to prolong the effect of epinephrin. There is no need to fear capillary bleeding three or four hours after its use. With the use of pituitrin it is possible to do more work in the office, without being afraid to send the patient home. The speaker, together with Dr. Kahn, recommended the use of this drug to the members as a preoperative procedure.

DR. A. M. CORWIN thought this a very interesting subject. In the modern use of some of the extracts of the ductless glands we have entered on a new era in therapeutics and physiology, and pituitrin seems to be coming into its own, as well as thyroid extract and others. This is partially due to its selective and safe action upon the unstriped muscles in the intestine, stimulating their contraction and their tone, and, owing to its affinity for the uterus, it bids fair to take the place of ergot. These things are all interesting to us. He remembered having read Citelli's article in the *Zeitschrift* last year, and while a few men have been working out the intimate problems of the physiology of this drug, there are comparatively few findings such as those presented by the essayists. They should be commended very highly, not only by internal recog-

*See page 322.

niton, but by external voice. This kind of careful analysis of a series of cases is done in too few instances, but it is the only way to arrive at sure results.

Dr. Corwin has used pituitrin in a number of cases. He has not had very many severe hemorrhages, but in those cases that he has been called in consultation to see since reading Citelli's first article, he has used this drug, and in several instances with very fascinating results.

With regard to the question of the invariable heightening of blood pressure, he was very much impressed with the case of a woman, fifty-five years of age, with well marked symptoms and findings of Bright's disease, in which there had been uncontrollable hemorrhage from the nose, an epistaxis that had defied packing and all sorts of things. This patient's blood was just like water, even thirty minutes or an hour after being drawn off. There was apparently no coagulation at all—or only a very faint sign of it. Blood pressure was two hundred. After very depleting hemorrhages her blood pressure had fallen, before the speaker saw her, to one hundred and sixty, and this had been maintained for a number of hours—perhaps six—in which the tests had been made. He injected pituitrin and the blood pressure came down within the next few hours, without any further hemorrhage to account for it, to one hundred and forty, and there it remained. In that case the urinary findings were those of a contracted kidney. Her pulse, however, became firm and strong, increased in force, and increased in strength, so that the cardiac stimulant was very apparent, and the question was whether this effect was due to the contraction of the vessels, which we are told occurs, except in those of the kidney, where dilatation takes place, causing diuresis.

Is the local effect due as in epinephrin, or also due to the selective affinity in the direction of blood coagulation, as in the various sera and antitoxins we inject, which we know produce the same sort of thing? This one case seemed to show him very clearly that pituitrin had this increased coagulating power, because within half an hour after the primary injection the effect was marked; the injections were repeated every six and then every twelve hours until the patient was entirely recovered.

Organic extract therapy is a revolutionary proposition. It

is simply marvelous, and he was very much pleased with the work of the essayists. However, the cases in this series were all children; another series must be studied in adults. It will probably be a number of years before we fully understand the true effects of this and the other extracts of tissues.

Just a word regarding the point of frequency of injections. In our work with the drug we give injections every six or twelve hours. It is arbitrary. Dr. Gordon's observation would seem to give us at least the opening door to this question, and twenty-four hours, perhaps, would seem to be often enough to make the injections.

DR. J. HOLINGER said that if the discussion was limited to pituitrin, he had nothing to say; but if the members would permit him to say a few words on stopping of hemorrhage, he would like to draw attention to a series of experiments published in the *Correspondenzblatt für Schweizer Aerzte* a few months ago, from the clinic of Professor Kocher, in Berlin. Fonia, an assistant of Professor Kocher, has studied the question of coagulation of blood, and found that the blood plaques cause the coagulation by means of a ferment contained in them and freed by their destruction. Therefore, he separates the plaques from the rest of the blood, and prepares them so that they keep. They form a brownish powder called coagulen. This is pressed against the wound by means of a piece of gauze and parenchymatous hemorrhage and small vessels stop bleeding at once. Coagulen is in the open market now.

DR. S. A. FRIEDBERG has been more concerned with the post-operative use of pituitrin. He has been using this drug at the County and Presbyterian hospitals for a little over a year, and his results have been uniformly good, with the exception of two cases, where there did not seem to be any beneficial result from its use. He operated on a case five days ago, and thus far this patient has required two injections, which have partly controlled the bleeding, but the patient is still expectorating blood. Dr. Friedberg admitted that there is a good field for the anteoperative use of pituitrin, and so far as the postoperative results are concerned, they have usually been good with him also.

DR. ROBERT SONNENSCHNIG said that during the last five or six months he has had pituitrin used as a prophylactic measure in practically every private case operated. It was used

fifteen to thirty minutes before operating, the dose being one-half cubic centimeter to children, and one cubic centimeter to adults. It would perhaps be unfair to say that it was merely coincidental, but in every case in which it has been used the hemorrhage has been very slight. In view of the fact that the administration of the drug carried with it practically no danger whatsoever, it would seem to him a good proposition to use it preliminary to operation. As Dr. Friedberg had said, in cases at the County Hospital the postoperative treatment has also been materially benefited.

DR. H. S. GRADLE said that the use of pituitrin cannot be limited to the practice of rhinologists alone. He used it in the gluteal muscle before resection of the tear sac, and did not think it was coincidental that he was able to make a very clean dissection, with practically no bleeding at all. He has never seen a tear sac operation where the field was so free from blood as in that case. In addition, he believes the drug will be found of value in operations for hemorrhagic glaucoma.

DR. OTTO J. STEIN asked if any of the cases reported had been followed long enough to know if there was any bleeding some time afterwards. That is, if there was any recurrence of hemorrhage. It would be of interest to know this. Following the use of many of these things, sometimes there is a bleeding that takes place some time afterwards. Also, he would like to know if the essayists had any particular experience with bleeding of other nature, not necessarily connected with any surgical work. Probably their experience has brought them into other fields of severe bleeding, coming on from various causes. These questions are of interest to all.

DR. WALKER asked Dr. Kahn if he had had any experience in cases of hemophilia or diabetes, in which there was liable to be a large amount of hemorrhage, such as an operation for adenoids or tonsils.

DR. NORVAL H. PIERCE asked Dr. Kahn if he has used atropin or scopolamin in connection with pituitrin. Have these drugs any influence on the pituitrin?

DR. E. H. M. GRIFFITHS said that pituitrin is his hobby. He has used it a great deal in abdominal surgery during the last four or five years. Speaking about hemorrhage in operations, he thinks that Cesarean section is the most bloody operation, but by the use of pituitrin he has been able to perform

it with very little loss of blood. He uses pituitrin in Cesarean section in the following way: One cubic centimeter is injected about half an hour before operation. At the time the patient is put on the table she is given another cubic centimeter, or fifteen minims. Immediately following the operation another cubic centimeter is given. This may seem an awful dose, but he does not think so. This is repeated at intervals of four to eight hours following the operation. When pituitrin was given in this way, there was practically no hemorrhage, temperature or shock; it was never necessary to catheterize the patient. However, he and his associates have been working on a different principle. They know that pituitrin does stimulate the involuntary muscles. Sajous claims that it acts by its stimulation of the adrenalin system, and that that is the reason for the rapid coagulation of the blood. Dr. Griffiths has also used pituitrin in other laparotomies, with wonderful results.

Regarding its effect on blood pressure, he has found where there is hypoadrenalism, pituitrin has increased the blood pressure. Where there is a decrease in adrenalism, it decreases it; and there are contraindications for the use of pituitrin.

DR. N. SCHOOLMAN has read Citelli's article in the *Zeitschrift*, and in that article pituitrin was reported as having been used in various cases for the control of hemorrhage, not only in operative cases, but also in subcutaneous hemorrhages, with good results—in hemoptysis and other hemorrhages. Citelli claims that it acts on the involuntary muscles, and its various manifestations throughout the system are explained thereby. He has found that it acts better while operating on an organ where the blood vessels have a well developed musculature, while in organs where there is not a well developed musculature its action is not so good. Therefore, he has said, in operating on the lower turbinate, where the musculature of the blood vessel is better developed, the constriction is greater and the effect is better than when operating on the septum. He asked if this observation was borne out by the essayists.

DR. KAHN replied in the affirmative.

DR. SCHOOLMAN said that the action of pituitrin, quoting Citelli, is similar but not at all identical to that of adrenalin,

and is different in that the action of adrenalin is rather intensive and rapid. It is followed also by very intensive and rapid reaction, and therefore the thrombus forming through the intensive, very short period of constriction is swept away by the rapid onwash of the blood during the reaction, whereas with pituitrin the action is slow and holds on for a long time. The reaction is correspondingly slow, and therefore the thrombus has had time to form, and the onrush of the blood following it has not such an impetus to sweep it aside.

Dr. Gordon had said that pituitrin increases the strength of the heart beat, and decreases its frequency. It undoubtedly does have a vasomotor constricting effect; it undoubtedly has an effect on the involuntary muscle cells.

The question of coagulability is a very interesting one. The speaker had mentioned the subject to a laboratory man, and he made the remark that maybe the base in which the pituitrin is dissolved has something to do with the coagulation. He thought that there was trikresol in most of the ampules. Whether there is anything in that or not, the speaker could not say. It might be well to try other solutions, or call it to the attention of the manufacturers.

Dr. GORDON, in closing the discussion on his part, said the remarks of the last speaker were rather more a discussion of Citelli's paper than the points brought out by Dr. Kahn and himself. Their desire had been to impress upon the members the coagulation power of pituitrin.

Regarding Dr. Holinger's remarks, he wished to reiterate Francini's statement. We have all been working with calcium salts, and have obtained more or less good from their use. Francini, in 1910, while studying body metabolism as influenced by the ingestion of the extract of the posterior lobe of the hypophysis, found that there was during its use a decrease of the magnesium and calcium salts in the tissues, while there was an increase of these salts in the blood stream. The speaker mentioned these facts to show how pituitrin probably acts.

Dr. KAHN, in closing the discussion, said that he hoped to continue this work, in association with Dr. Gordon, and bring a further report to the society.

As to Dr. Holinger's remark about fifteen minutes' wait, of course, when you have severe hemorrhage, waiting fifteen minutes for the effect of a drug is important. But pituitary

extract can be used as a prophylactic in operative procedures in those cases where hemorrhage is expected, and thus the difficulty overcome. The speaker did not claim that the drug is a cure-all. You may get cases in which you will have to use blood serum following pituitrin. They did not claim that it was infallible. They only claimed that the blood does coagulate more rapidly under pituitrin.

As to the use of the drug in hemophilia and diabetes, he could not report about that at this time. His own experience with hemophilia has been limited to only one near hemophilic case, but Dr. Gordon had one case in which the coagulation time was fifteen minutes, brought down to three minutes. Those are the only cases of hemophilia in which he knows that the drug was used. As to its use in diabetes, he knows nothing of it.

Answering Dr. Pierce's question, he has never used atropin or scopolamin with pituitrin.

Paper: Tuberculosis of the Larynx.*

By J. HOLINGER, M. D.,

CHICAGO.

DISCUSSION.

DR. O. J. STEIN wanted to take exception to the statement that external surgical procedures—and the essayist mentioned laryngofissure—are objectionable. He feels that there is a field for this operation in certain forms of localized tuberculosis of the larynx. Without going into details, his experience has led him to feel that well defined local tumefactions of a tubercular character can be eradicated, and the probabilities of a dissemination of the disease in the larynx, or possibly elsewhere, can be mitigated.

In this connection, he wanted to ask if any of the members had had any experience in the use of tuberculin locally in the larynx. He did not think that was mentioned in the paper. There has been a good deal of work done, especially by men who are doing a great deal of work with tuberculosis, as those in Colorado and California. The application of tuberculin, especially the old Koch tuberculin, upon the diseased tissues

*See page 327.

has led these men to make some reports of very striking results. He has tried it a few times, but was not in a position to give a report of them at this time.

Dr. E. P. NORCROSS thinks there is a very pessimistic opinion held as to the curability of laryngeal tuberculosis. This may be due to the great variation in statistics dealing with the frequency of this complication of pulmonary tuberculosis. The reason for this variation is that cases are not diagnosed early. We wait until the patient complains of a little hoarseness or dryness in the throat, or, if they are examined, the changes are not recognized, and possibly the pulmonary end is so intense that it overshadows the laryngeal. The speaker thinks that if the cases were all examined carefully, in the same way that we are taught to recognize early changes in the lung, the percentage of cases of consumptives having tubercular laryngitis would be greatly augmented, and naturally, then, the percentage of cures would also be augmented. One cannot help but believe that there are many cases of tubercular laryngitis never diagnosed that heal spontaneously, just the same as the lung trouble may heal. His point is this: If all the consumptive patients were carefully examined regarding the condition of the larynx, the percentage having this involvement would be great, and the cures would naturally rise also.

Dr. O. T. FREER agreed with Dr. Holinger concerning the uselessness of external operations in most cases of laryngeal tuberculosis. This is the conclusion of Dr. Nils Arnoldson, of Stockholm, who has made an exhaustive study of the matter. He says (*Archiv für Laryngologie*, 1913, page 46): "In the literature of later years little belief is expressed in the possibility of material benefit from the external operations, least of all from total extirpation of the larynx." Gluck, quoted in Arnoldson's article, however, advises external operation where all of the tubercular disease may be extirpated by it, and states that in many cases laryngeal tuberculosis is merely an active focus of localized tuberculosis.

Undue optimism in regard to recoveries obtained by endolaryngeal surgery must also be avoided. Where a tubercular focus may be excised with uninfected tissue about it, as in the case of tuberculosis of the epiglottis confined to the periphery of its free pharyngeal portion, a permanent cure may be ob-

tained. But where the disease has entered into the tissues of the body of the larynx itself, as in ulcers of the cords, aryepiglottic folds, ventricular bands, and especially where there is tubercular, infiltrative chronic edema or perichondritis, complete extirpation by endolaryngeal cutting operations is impossible, for there are always miliary foci scattered far about the main seat of the disease, which lie in apparently healthy tissue, and which reproduce the tubercular process. This may be seen especially well in nasal tuberculosis, where an apparent complete removal of the tubercular focus is so easy, for in spite of such removal a return of the disease is the rule.

Dr. Freer agreed emphatically with Dr. Holinger that laryngeal tuberculosis is often primary. There never has been proof advanced for the often repeated assertion in the textbooks that laryngeal tuberculosis always follows pulmonary tuberculosis, and is evidence that a pulmonary focus exists. Dr. Freer always examines the chest in cases of laryngeal tuberculosis, and has often, even in advanced cases, failed to find any sign of pulmonary disease. To assert that, nevertheless, a primary pulmonary focus must be there simply because there is laryngeal tuberculosis, is merely an unproved, dogmatic statement. Later, of course, the lung nearly always becomes infected with tuberculosis in the form of aspiration tuberculosis.

DR. L. E. GORDON wished to subscribe to what the essayist said, but would include nasal troubles. The essayist mentioned pharyngitis, but not the nasal affections that may be found in these cases. Dr. Gordon's experience has led him to the conclusion that the larynx is not such a vulnerable part of the body, and would be able to overcome tubercle bacilli invasions from the lungs if the upper respiratory passages were normal. His experience in operative procedures upon tubercular patients has been limited to those for correction of deformities in the nose and throat. He has seen a number of cases that were on the sanatorium waiting list clear up after degenerated tonsils were taken out, deformities in the septum or turbinal bones corrected, and this observation has led him to the conclusion that the larynx is not very susceptible to invasion from the lungs, provided the upper respiratory passages are normal.

Dr. E. L. KENYON wanted to say just a word in confirmation of Dr. Norcross' position, and to go to a little further. He is inclined to think that the laryngologist does not apply the same intelligence to the examination of these cases of tubercular laryngitis as the internist does to the pulmonary condition. The attitude of the physician treating pulmonary tuberculosis is largely the attitude of prophylaxis. He says: Attack the tuberculosis of the lungs before the lungs are attacked with the tuberculosis, as it were. In other words, so treat your patient as to increase his resistance. The speaker believes that laryngeal tuberculosis as a primary proposition is extremely rare, because he believes that the conditions necessary to produce tubercular laryngitis are exceedingly rare, excepting in connection with pulmonary tuberculosis. In cases of pulmonary tuberculosis, one is able to watch the development of laryngeal tuberculosis if he watches the larynx. Not only that, but he is able to see the conditions in the larynx which predispose to pulmonary tuberculosis. He sees the local laryngitis which is tending to result in abrasions of the mucous membrane, in which abrasions the tubercle bacilli become engrafted. The time will come, in the opinion of the speaker, when laryngologists will examine every pulmonary tuberculous patient systematically for trouble in the larynx, not once in six months, or once in a long period, but as an essential part of the treatment of the pulmonary condition as frequently as necessary. Watch will be kept of the larynx, and primary disturbances will be treated with the purpose of avoiding laryngeal tuberculosis. That will be the advanced future attitude of the laryngologist in reference to laryngeal tuberculosis.

Dr. Kenyon has recently come to feel that the development of laryngeal tuberculosis is dependent more than we have heretofore thought upon cough. He is coming more and more to feel that cough is a primal factor in the development of laryngeal tuberculosis, because cough involves the most violent action we have in the larynx, and it is this violent action in the larynx, taken with a chronic laryngitis, that he feels is responsible to a great extent for the infection of the larynx with the tubercle bacillus. In the act of coughing the breath is taken in, the vocal cords are jammed together, and are held tightly, while the chest is contracted on the air column in the

bronchi and trachea. The vocal cords must be held so tightly that the air does not pass through them with the contracting chest. Then the larynx is quickly opened and the air bursts through. In that process of coughing the vocal cords are not gently held, as in talking, but are held so tightly that it is possible to produce an abrasion of the cord and thus encourage the infection to find lodgment upon the cord. Likewise, the movement of the muscles in the arytenoid region are more violent in coughing than in talking. Here, owing in cases of pulmonary tuberculosis to the almost universal presence of infiltration, the flexibility of the mucous membrane has become reduced, and abrasions in which tubercle bacilli may find lodgment are likely to be produced. So the speaker thinks that this violent action of coughing may explain a good deal of laryngeal tuberculosis.

DR. H. KAHN said that one point to be considered in the surgical interference in laryngeal tuberculosis is whether the condition is one of open or closed tuberculosis. Surgical interference in laryngeal tuberculosis where there is temperature is absolutely contraindicated. That is the real crux of the situation. When temperature is present, only palliative measures are indicated.

Regarding external operations, Hajek, in the *Medicinische Klinik*, 1912, page 385, has reported excellent results with tracheotomy. The larynx is placed absolutely at rest for a period of some length, and most of the cases recovered.

There is no dearth of sanatoria for the treatment of the indigent tuberculous in Chicago. Oak Forest has now over six hundred patients, and will soon have room for two hundred more. The Chicago Tuberculosis Sanatorium will soon open up with nearly a thousand beds, so that the poor can have all the necessary treatment. There are many other sanatoria for the middle class, who are able to pay something for their treatment.

DR. HOLINGER, in closing the discussion, said that he knew that his standpoint on the question of laryngofissure was not shared by all members, but all his cases operated by laryngofissure did not do at all well. The impression he received was that the excitement of the operation and after-treatment pulled the patient down.

The speaker noticed that he had been misunderstood by

Dr. Norcross. He said that, on the whole, the prognosis of laryngeal tuberculosis is good. The main point is early diagnosis. Dr. Norcross spoke about early examinations—not only early, but regular examinations—of the larynx of consumptives. The speaker has been many times in a very disagreeable position, when patients suffering from tuberculosis of the lungs came and complained about their larynx. Their physician contended that the hoarseness, etc., amounted to nothing, and did not need special attention. Add to that the changeability of the symptoms, the patients sometimes having absolutely no symptoms for weeks. What should the specialist say and do when suddenly bad laryngeal symptoms appear? Is it his duty then to protect the general practitioner before the patient? How is he going to justify serious therapeutic measures before the patient?

As to the frequency of diseases of the nose in tuberculosis of the lungs, it is textbook knowledge that tuberculosis of the lungs and larynx is three and four times more frequent in patients who have irregularities in the nose than in those with normal noses.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting Held December 22, 1914.

DR. OTTO J. STEIN, THE PRESIDENT, IN THE CHAIR.

Probable Syphilitic Ulceration of Left Tonsil.

DR. W. G. HATCH, Rockford, in presenting this case, said that the family history was absolutely negative. About six months ago the patient complained of trouble with the throat, accompanied by swelling, at which time the speaker first saw him. There was then a very large ulceration of the left tonsil present. The glands were very hard and indurated on both sides. He did not feel that the case was operative, and felt sure that it was malignant. He did not want to make a section, because he did not wish to subject the patient to the dangers of metastatic infection. He did not have a Wassermann made, because his experience with this test has not been particularly happy. He put the patient on treatment with iodids, and the condition commenced to clear up. The glands are getting smaller and the ulcers seem to be healing. The speaker was inclined to think the condition was due to syphilis.

DISCUSSION.

DR. NORVAL H. PIERCE believes all such cases as the one presented should be subjected to microscopic examination immediately. The danger of opening up the lymphatics by operating to take out a piece of such tissue is very slight; in fact, hardly exists. If we wish to seal up the lymphatics immediately afterwards, we can seal over the wound with the cauter, and this will obviate any danger that the doctor who reported the case seemed to believe exists. It is not a matter of whether a case is operable or not; it is a matter of making a diagnosis. There are several things that come into thought at this point, as to whether it can be carcinoma or actinomycosis or syphilis—even tuberculosis, although, of course,

it does not have that appearance; yet tuberculosis in exceptional instances may so very closely simulate carcinoma that the differential diagnosis can only be made by microscopic examination. If the diagnosis of syphilis is made positively, one is very much more apt to be a little more heroic in treatment. The case does not seem to be inoperable now, although the involvement of the glands on both sides of the neck would seem to indicate that the process had gone to a point where the issue of an operation would be very doubtful.

However, the point on which he wished to be understood was that all these cases should be immediately subjected to microscopic examination. The danger from taking out a piece of such a growth is not at all great; in fact, it should not stand in the way for a moment of making the diagnosis. If we wish to seal up the lymphatics, we can do so with the actual cautery, so that danger is altogether done away with.

Carcinoma of Upper Jaw, With Resection.

DR. JOSEPH C. BECK showed a patient who had had a carcinoma of the left upper jaw, which the speaker diagnosed by microscopic examination. It extended somewhat on the anterior surface of the superior maxilla when seen, as the condition had lasted for eight months. He wished especially to refer to the operation in this case, particularly the technic. A resection of the upper jaw without external incision was made, as in an antrum operation. He excised the inner surface of the cheek wide of the tumor. Then, internally, in the midline, below the upper lip, just under the nose, raising a flap as in the Loewy operation, he resected the superior maxilla posteriorly, including the palatal bone. It was not necessary in this case to remove the lateral wall of the nose, or the floor of the orbit, because they were not involved. The carotid artery was not ligated at the beginning of the operation, but on account of a good deal of bleeding a temporary compression, according to the Crile method, was performed, which resulted in practically no bleeding during the remainder of the operation. After the operation was completed, small particles of the tissue from the operative field were excised and saved for microscopic examination. This tissue was found

subsequently absolutely free from carcinoma. Then a layer of gauze was placed within the cavity, and on top of that ten milligrams of radium element, some more gauze, and the soft palate sutured temporarily to the side of the cheek to control bleeding. The radium was allowed to remain in for forty-eight hours. The gauze was removed next day and replaced three times within a week, and then left out. Since then the sloughs have been coming away, and there is still a portion of the slough left in the anterior wall. It is now four weeks since operation, and the patient was shown because he was leaving for his home next week. Perhaps it would be of interest to see him some time in the future with no recurrence.

In this connection, Dr. Beck showed a stereoscopic photograph of a patient operated on seven years ago by the external method, and the patient is alive and in good health at the present time. The operation, however, leaves quite a different face from what the patient just exhibited has. Dr. Beck believes that, as a rule, the internal operation can be very nicely done in these cases. He employed the burr almost exclusively in the removal of the bone, and it was very easy to manipulate. Dr. Beck expects to have a prosthesis made with an artificial denture attached, but this must not be worn until all postoperative reaction has disappeared.

Tuberculosis of Pharynx and Soft Palate.

DR. J. HOLINGER presented two cases. The first patient came to Alexian Brothers Hospital complaining of indefinite throat trouble. On examination, there was an area just in front of the right tonsil, about the size of a quarter, swollen, and of irregular surface. Pieces were removed for examination which showed nothing indicative of syphilis or carcinoma. The process progressed very fast, and in about two weeks the whole half of the soft palate was involved, in another week the whole of the soft palate and a part of the hard palate. The uvula was eaten out in characteristic manner. Pieces removed at this time for examination showed typical tubercles. At the same time word came from Dr. Rettig, the internist, that the lungs had râles all over. In the sputum tubercle bacilli were found. The man at that time looked very robust and healthy, which appearance he has lost. The

whole soft palate, pharynx and epiglottis are eaten out by the tubercular process. It extends up into the nasopharynx and down into the larynx and esophagus, as far as we can see. Other isolated foci are on the inside of the lower lip. This is not by any means a frequent condition. In the *Centralblatt für Laryngologie* this year there have been four or five cases mentioned of tuberculosis of the pharynx, all of them developing very slowly, not progressing in many months as far as this one had in weeks.

Rhinoscleroma.

This patient came to Alexian Brothers Hospital with both nostrils absolutely closed. Dr. Holinger could not make the slightest impression with the probe on either the turbinals or septum. The septum was equally swollen on both sides, as were the turbinals. The cocaine did not shrink the swellings. The speaker forced some packs of cotton with mercury salve into the nose, repeating that every day for many weeks, and with this treatment the swelling of the septum and turbinals went down some, and at the present time the nose is comparatively free. Several large pieces were cut from the septum, but unfortunately were stained wrongly, and the speaker would have to wait for a few days longer before getting a report. If, as the speaker thinks, it is a case of rhinoscleroma, it can be only the beginning, because the trouble only dates back to last summer. The patient is from Ruthenia. The geography is, as the members know, of importance in rhinoscleroma.

DISCUSSION.

DR. S. A. FRIEDBERG asked Dr. Holinger if there was anything characteristic on palpation of the nose in the case of supposed rhinoscleroma.

DR. HOLINGER replied that the nose was rigid, and could not be compressed at all.

DR. FRIEDBERG said he had so far seen three cases of rhinoscleroma at the County Hospital, and at the present time has one under observation that has been in the hospital for a couple of years. One case which he recalled had practically the same characteristics as the one presented by Dr. Holinger. The case to which Dr. Friedberg referred had a combined

nasal and laryngeal involvement. The larynx showed the typical subglottic swelling. It was necessary to perform a tracheotomy and the man wore a tube for over a year. After the tracheotomy tube was removed, an intubation tube was worn for a short time. At present the patient is without any tube, and is getting along nicely. The scars can still be seen in the larynx, but there does not seem to be any progression.

Regarding the bacteriologic findings in these cases, we know that we have an encapsulated bacillus resembling the Friedlander bacillus to a great extent, and for that reason some doubt has been cast on the fact as to whether this is the absolute etiologic factor. The speaker thinks more is to be demonstrated from the sections. If both the nose and larynx are involved, it is usually easy to make the diagnosis in a good many of the cases. It would be interesting to see what the specimens would show.

DR. GEORGE W. BOOT, referring to the first case shown by Dr. Holinger, of tuberculosis of the pharynx, said that not long ago he saw a woman with what he believed was tuberculosis of the pharynx. She gave a history of having had trouble for twenty years, which had been diagnosed as syphilis at various times, but treatment along antisiphilic lines did no good. The speaker considered the case one of tuberculosis of the pharynx, and ordered very small doses of tuberculin once a week. At the end of about two months the throat is feeling better than for a long time, and looking better. The process has healed over, with the exception of a small ulcer on the right side. She has gained many pounds in flesh under this treatment. The Wassermann was negative.

Carcinoma of the Throat Involving the Esophagus. Miliary Tuberculosis.

DR. GEORGE W. BOOT exhibited, first, a specimen from a carcinoma of the throat, involving the esophagus, left side of the pharynx and left side of larynx.

Second, larynx from a case of miliary tuberculosis. This patient, a man, came to Cook County Hospital suffering from a severe laryngitis, so that the speaker could not get a satisfactory view of the larynx. He did not return to the hospital for three or four days, and in the meantime the man had

died. At the last he had a great deal of dyspnea, so that the internes did a tracheotomy, rather high, cutting off the cricoid cartilage. At the postmortem there was found tuberculosis of the lungs and larynx. There was a shallow ulcer on the epiglottis, and below the vocal cords there was an ulcer on either side, with quite a number of miliary tubercles.

Bony Tumor Filling Nasopharynx, With Exhibition of Photograph.

DR. L. W. DEAN showed a photograph of a bony tumor that completely filled the nasopharynx. The soft palate was not involved, but it was pressed downward and forward, so that the tumor could be seen in the nasopharynx. The tumor could be felt as a hard bony mass. Entrance could not be made into the nasopharynx, in order to tell just where the tumor grew from.

Operation.—Tracheotomy; anterior posterior incision in the midline of the roof of the mouth, severing the soft tissues down to the bone, and the soft palate to the tip of the uvula. These tissues were retracted laterally and the posterior third of the hard palate removed. The growth was found growing from the upper surface of the hard palate and from the posterior surface of the vomer. The palatal wound was closed by Brophy's method of closing posterior cleft palate. The closure was complete. Speech perfect.

The microscopic diagnosis was osteoma. Six months later there was no recurrence.

DISCUSSION.

DR. OTO J. STEIN asked how hard the bone was.

DR. DEAN replied that it was not ivory-like; it was a crackling affair.

DR. STEIN asked if the nose was involved.

DR. DEAN replied, not at all.

DR. STEIN said that, as a rule, osteomata of the nose are extremely hard.

Surgical Anatomy of Temporal Bone, With Lantern Demonstration.

DR. GEORGE E. SHAMBAUGH stated that every otologist of today aims to be an aural surgeon, but that this was not the case even a few years ago. During the earlier days of the

specialty of otology the complications resulting from mastoiditis were usually taken care of by a general surgeon. This state of affairs has largely disappeared in this country, and there are very few first-class general surgeons who are still willing to undertake the special surgery of the ear. Some of the general surgeons who have not kept in touch with the advances of modern otology are still unaware that the type of operation which the specialists are able to do for the cure of chronic, as well as acute, mastoiditis is something quite different from what the general surgeon is prepared to undertake. In England it is still not uncommon for some general surgeons to do mastoid work; indeed, some valuable contributions to the surgery of the mastoid have been made by general surgeons in England, who have devoted a good deal of their energy to the question of mastoid surgery. In Germany it seems that mastoid surgery has been taken over entirely by the aural surgeons, although as late as fifteen or twenty years ago the general surgeons still did a large amount of the mastoid surgery that was being done.

Everywhere at present we have come to realize that the complications which arise from suppurations of the middle ear, including mastoid disease, labyrinth infection, and the intracranial complications, are taken care of more efficiently by the aural surgeon than by men engaged in general surgery. This means, of course, that the specialist in otology must master the details of the regional anatomy of the ear, in order to do this work properly.

As a matter of fact, there is only a small proportion of ear cases where surgical interference is necessary, and yet every otologist feels, and rightly so, that he should be prepared to take care of those complications which develop in the course of suppurative middle ear disease. In order to be prepared to do this work the aurist must be trained not only to handle a chisel in a dexterous way, and to ligate vessels, but it is absolutely necessary that he should master the regional anatomy of the ear. This is not such an easy thing to do, as many of the men in the audience could testify. The conditions presented by pathologic changes in the mastoid make it necessary for one to have a perfectly clear mental picture of the various relations, if one is to operate without injuring important

anatomic structures. Surgical anatomy of the ear can never be acquired from textbooks or descriptions. One might read all of the descriptions written on the anatomy of the ear and study all of the drawings that have been made of this region, and yet, if he has not actually handled anatomic preparations showing the relations, he will find himself very inadequately prepared when attempting to operate on the ear. The method usually employed in gaining this anatomic knowledge is by beginning to do the operations on the cadaver. This, however, is not the best way to acquire a mental picture of the anatomic relations of the mastoid, for the reason that most of the relations are destroyed while attempting to expose this or that structure. One may operate upon a great many different temporal bones in this way and still have a very hazy idea of the relations of the various structures. The best way of acquiring a mental picture of the anatomy of the ear is by studying sections made through the temporal bone, each section devised so as to bring out clearly certain definite relations. Sections made at random through the temporal bone will be of very little assistance. The speaker recalled looking over, a few years ago, a series of sections made in this way. He was surprised to see how few of the sections brought out relations which were of any practical value. The variety of preparations which one can make of the temporal bone is very great. The speaker spent a great deal of time several years ago in working out a set of preparations, each devised so as to bring out certain definite relations. In all he made something over fifty different preparations. In the lantern demonstration which was to follow he would show some of these preparations.

In closing his introductory remarks, the speaker called attention to the accidents which the general surgeons have so frequently had when operating on the ear; accidents which the carefully prepared aural surgeon very rarely has. An injury to the facial nerve by an aural surgeon is now extremely rare. It is the impression of the speaker that the amount of aural surgery done today is less than what was done fifteen or twenty years ago. He attributes two reasons for this: First, that the suppurative diseases of the middle ear are better taken care of now, since the public recognizes the danger in

running ears; the other reason is that the aurists are able to distinguish much more definitely among the cases of chronic suppurative middle ear trouble those where the danger of a complication is imminent, and those where the danger is very remote. The percentage of cases where in chronic purulent otitis media an operation is indicated is relatively small. There was a time, after the danger from chronic running ears became recognized, and when the differentiation was still hazy between cases which were likely to have complications and those which were not, that otologists were operating on a good many of the nondangerous type. This period has now passed, and a careful examination of the ear readily distinguishes, as a rule, between cases which require a radical mastoid and the more common type of chronic running ears where nothing more than the local treatment need be carried out.

Paper: Hammond's Mastoid Skin Grafting—A Preliminary Report.*

BY FRANK ALLPORT, M. D., AND ALEX. S. ROCHESTER, M. D.,
CHICAGO.

DISCUSSION ON THE PAPERS OF DR. SHAMBAUGH AND DR. ALLPORT.

DR. NORVAL H. PIERCE asked Dr. Allport if these grafts to which he referred were all primary grafts, to which Dr. Allport replied yes.

DR. J. HOLINGER wished to support Dr. Shambaugh emphatically in his statement that the otologist ought to operate these cases, not the general surgeon, and hoped Dr. Shambaugh could find a way to incorporate this statement into the laws of ethics of the profession. He wished to emphasize this fact, because the present condition is an evident injustice to otologists. It lowers them to the position of routine treaters of running ears. It effaces a great deal of enthusiasm if the surgeon takes away the ideally and financially interesting work. We have all seen the results of surgeons operating on ears in the form of facial paralysis, destroyed hearing, etc., referred to by Dr. Shambaugh.

With reference to the planes in which temporal bones should be cut for studying the anatomy, he wished to refer to one

*See page 335.

plane of cutting which gives much more information than others, namely, the plane parallel to the tympanic cavity and through the eustachian tube. It takes quite a good deal of thinking in order to bring that plane into position. You have to open the middle ear through the tegmen tympani and study accurately the position of the saw before you start cutting, but when you succeed in splitting the eustachian tube in half and at the same time not injuring the tympanic membrane, you will be surprised how clearly you see the position of the aditus ad antrum, of the antrum, and of the facial nerve relative to the parts in the mastoid process, and relative to the labyrinth.

Regarding Dr. Allport's paper, he wished to remind him that every one of the points brought out are set down in the paper of Siebenmann, in the first number of the *Berliner klinische Wochenschrift*, in 1893. What Ballance has to say about secondary grafting can all be found there. The French and English authors at that time not only criticized, but actually laughed at these endeavors of the German and Swiss authors in dealing with chronic suppurations. The speaker heard the ridicule that was manifested against all these points when he was in Paris. Ballance, and others referred to by Dr. Allport, came long afterwards, and Dr. Holinger does not think it just that they should get credit for things that were brought out many years before they considered the matter. It ought to be a scientific principle to give credit to whom it is due.

The speaker has done a good deal of secondary skin grafting—as early as 1893. Not long ago he saw the first patient he operated upon in May, 1893. The ear was still dry, and the hearing good.

DR. GEORGE W. BOOT had been very much interested in the lantern slide demonstration of Dr. Shambaugh. A few years ago he saw a mastoid in an infant where the bulging of a subperiosteal abscess was in the fossa triangularis. The infant was about seven days old. There had been no discharge from the external auditory canal.

DR. JOSEPH C. BECK said that he thought if Dr. Holinger would give an outline of how the section he referred to is made, probably some of the men present would make such sections

for themselves. Piffl, of Prague, had shown Dr. Beck how to make them by taking a line from the tip of the petrous portion of the temporal bone as one landmark, and the tip of the mastoid as the other, sawing through the petrous portion of the temporal bone. In this way you get that section just about right, and you will find that in making a number of these sections you will just about get the outline that Dr. Holinger mentioned. However, the speaker doubts very much whether you could get the information from only that one section, as from those shown by Dr. Shambaugh. He felt very much indebted to Dr. Shambaugh for his presentation. While it was perhaps not exactly new, yet it is most important for us to rehearse and see these specimens.

Regarding Dr. Allport's paper, no matter who has the right to priority in Germany, at least, so far as the speaker knows, Ballance is the man who has made skin grafting in mastoids the operation it is at the present time. Perhaps it is the fault of the readers—that they do not read so much of the German literature. At any rate, it is the excellent work of Ballance we are always connecting with our work in mastoid surgery. That does not mean that we would like to take anything away from Germany.

Dr. Beck has employed skin grafts ever since he learned to do the radical mastoid operation, using them, giving them up, and taking them up again, doing them in every way suggested, and when Dr. Hammond showed his method at the Otological Society, he tried his method, and gave it up, and is now using the primary skin graft. After smoothing the bone of the mastoid cavity as much as possible, immediately placing large grafts—two, and at most three, in number. He has never scraped or burred the inner wall of the middle ear, or any part therein. He believes that to be unnecessary. It is very dangerous, and he does not think it should be done. The speaker felt the members were indebted to Dr. Allport for his exposition of the subject, even if it is old.

DR. ALLPORT, closing, said that he did not intend to present anything new to the society. There is certainly nothing new in skin grafting, and his object had merely been to give a résumé of the subject.

Paper: Choanal Fibroma.*

BY NORVAL H. PIERCE, M. D.,

CHICAGO.

DISCUSSION.

DR. GEORGE W. BOOT wished to show three specimens in connection with Dr. Pierce's paper, the first of which he thought would fall in Dr. Pierce's classification. It was removed from the left nostril of a man, about thirty-eight years of age. The growth could be seen in the nasopharynx. It involved some of the anterior ethmoidal cells, and came from the maxillary sinus. It was removed by evulsion and taken out through the nose. Sections showed it to be a fibrous polyp.

The second specimen was that of a postnasal tumor, which, when removed, the speaker thought was a fibroma, but on cutting the sections it proved to be a fibrochondroosteoma. It was attached to the base of the cranium, and the speaker experienced great difficulty in getting it out. No. 8 wire in the snare failed to make any impression. Finally, he cut it loose from the base of the skull with the forceps, and finished the dissection with Killian's bent knife.

The third specimen was one which was removed from a child, three and one-half years old. The growth could be seen through the mouth, and the speaker thought it was a case of adenoids. After removal, section showed it to be a sarcoma. The child had a very prompt recurrence, was taken from the hospital, and died.

DR. OTTO J. STEIN has always been under the impression that choanal fibromas had one attachment, as stated by the essayist. He had a case some years ago that he operated, and at that time thought there were two attachments. He presented the specimen—a large one, about two and a half to three inches long—to the society, but in discussion (at St. Louis) the members finally convinced him that one of the attachments was nothing but an adhesion that had taken place. Dr. Pierce said that one of his cases had an adhesion, but

*See page 332.

he also stated that he had one that had no adhesion, and asked Dr. Pierce if that was not what he had said.

DR. PIERCE replied that the case referred to had no adhesions. It is said that these tumors do not form adhesions, as a rule.

DR. STEIN, therefore, still had some hope that his case did have two attachments instead of one and an adhesion, as the members had convinced him at that time.

